# Routine Troponin Measurements Are Unnecessary to Exclude Asymptomatic Coronary Events in Acute Ischemic Stroke Patients

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*Background:* Obtaining serum troponin levels in every patient with acute stroke is recommended in recent stroke guidelines, but there is no evidence that these contribute positively to clinical care. We sought to determine the clinical significance of measuring troponin levels in acute ischemic stroke patients. *Methods:* We reviewed 398 consecutive patients with acute ischemic stroke at a large academic institution from 2010 to 2012. Troponin levels were measured as a result of protocol in place during part of the study period. The mean age was 70 years (standard deviation ±16 years) and 197 (49.5%) were men. *Results:* Chronic kidney disease was present in 78 (19.6%), coronary artery disease in 107 (26.9%), and atrial fibrillation in 107 (26.9%). Serum troponin T was measured in 246 of 398 patients (61.8%). Troponin was elevated (>.01 ng/mL) at any point in 38 of 246 patients (15.5%) and was elevated in 28 patients at all 3 measurements (11.3% of those with troponin measured). Only 4 of 246 patients (1.6%) had a significant uptrend. Two were iatrogenic in the setting of hemodynamic augmentation using vasopressors to maintain cerebral perfusion. One case was attributed to stroke and chronic kidney disease and another case to heart failure from inflammatory fibrocalcific mitral valvular heart disease. *Conclusions:* Serum troponin elevation in patients with ischemic stroke is not usually caused by clinically significant acute myocardial ischemia unless iatrogenic in the setting of vasopressor administration. Serum troponin levels should be measured judicially, based on clinical context, rather than routinely in all stroke patients. **Key Words:** Acute stroke—troponin—ischemic stroke—coronary events.


## Introduction

Elevations in serum cardiac biomarkers—in combination with clinical symptoms and electrocardiographic findings—are essential to the diagnosis and management of acute myocardial infarction (AMI).[1,2] Cardiac troponin I and cardiac troponin T (cTnT) are more specific and more sensitive than creatine kinase myocardial b (MB) fraction measurements and are thus preferred.[2] However, clinicians overuse cardiac biomarkers in clinical practice, even in evaluating patients with suspected cardiac disease.[3]

Although elevated troponin levels are fairly specific for AMI, they may be caused by other conditions including congestive heart failure, left ventricular hypertrophy, chronic kidney disease, and diabetes mellitus.[4] Elevated troponin levels have also been found in 10%-20% of patients with acute ischemic stroke.[5,6] The clinical significance of raised troponin levels—and thus the utility of measuring them—in acute stroke patients is uncertain. Some have

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questioned whether serum troponin should be routinely measured in this population.[7] Results of tests that are not clinically indicated may be financially wasteful, time-consuming, difficult to interpret, and distracting from a diagnostic standpoint. Furthermore, they can lead to unnecessary testing and unwarranted patient concern. Yet current stroke guidelines by the American Heart and Stroke Association recommend measuring troponin levels in all ischemic stroke patients.[8]

Supporting evidence suggests troponins add prognostic significance.[9-11] However, many stroke physicians do not rely on troponin levels for prognostication, and whether routinely obtaining serum troponin in stroke patients influences acute clinical management remains unclear.

In the present study, we aimed to evaluate the frequency and pattern of serum troponin elevation in acute ischemic stroke patients and to determine the impact on clinical practice.

## Methods

We retrospectively reviewed 398 consecutive adult patients with acute ischemic stroke admitted to the Mayo Clinic Hospital, Saint Mary's Campus, from 2010 to 2012. These patients were prospectively collected for the American Heart and Stroke Association's Get With The Guidelines Stroke Registry. Patients with stroke due to intracranial hemorrhage, subarachnoid hemorrhage, or subdural hematomas were excluded. Only patients with acute ischemic stroke listed as the primary/principal diagnosis were included. The following variables were collected: age, gender, weight, National Institutes of Health Stroke Scale (NIHSS) score at admission, serum creatinine level, medical comorbidities (coronary artery disease, atrial fibrillation, hypertension, diabetes mellitus, dyslipidemia, and chronic kidney disease), timing and results of serum troponin levels, whether the cardiology service was consulted, and what the results of the electrocardiogram (ECG) and echocardiogram revealed, and when performed.

Serum cTnT levels were ordered and drawn in the emergency department or immediately after hospital admission. Our institution's protocol included routine troponin measurements in all stroke patients until 2011. Therefore, some patients within the study time period included did not have troponin levels. cTnT levels were analyzed by the Mayo Clinic Laboratory using the Cobas e411 method. Values greater than .01 ng/mL were considered elevated. A rise of greater than 20% between the 0-, 3-, and 6-hour values was considered a significant delta.[12]

The Stop Stroke Study–Trial of Org 10172 in Acute Stroke Treatment classification was used to characterize stroke etiology: large-artery atherosclerosis, cardioaortic embolism, artery occlusion, undetermined causes, and other mechanisms (e.g., dissection, cardiac or arterial surgery, intervention).[13,14] Functional outcome was recorded at the time of hospital discharge using the modified Rankin Scale (mRS), which consists of 6 grades: 0, no symptoms; 1, no significant disability with symptoms; 2, slight disability; 3, moderate disability; 4, moderately severe disability; 5, severe disability; and 6, dead. ECGs were interpreted by Mayo Clinic cardiologists and the published reports in the electronic medical record were used for the purposes of the current study. We also collected data on stroke location based on neuroradiology reports. Data analysis was performed using the JMP software (developed by Statistical Analysis System in Cary NC, USA). The present study was approved by our institutional review board.

## Results

Of 398 patients, approximately half (n = 197, 49.5%) were male. The mean age was 70 years (standard deviation ±16 years). The median NIHSS score at admission was 4 (interquartile range [IQR] 2-8). Half of the patients (n = 199, 50.0%) had strokes involving the right hemisphere. There was a widespread distribution of stroke mechanisms observed in our patients. Cardioembolic stroke was present in 101 (25.3%), large-artery occlusion in 122 (30.6%), small-artery occlusion in 84 (21.1%), undetermined causes in 87 (21.8%), and other mechanisms in 4 (1.0%). Intravenous recombinant tissue plasminogen activator was administered in 49 patients (12.3%).

Table 1 displays baseline patient characteristics, including a comparison of patients with and without serum troponin measurements. Patients whose troponins were measured tended to be older, had higher NIHSS scores on admission, and had a greater frequency of dyslipidemia.

Serum cTnT level was measured in 246 patients (61.8%). A single level was obtained in 58 patients (23.6% of those with troponin measured), while serial measurements at 0, 3, and 6 hours were available for 188 patients (76.4% of those with troponin measured). Serum troponin level was elevated at any time point in 38 of 246 patients (15.5%). Thirty-two patients had an elevated troponin reading at the 0-hour measurement (median .05 ng/mL [IQR .04-.08]); at 3 hours, 28 patients had an elevated reading (median .05 ng/mL [IQR .022-.11]); and at 6 hours, 34 patients had an elevated troponin reading (median .06 ng/mL [IQR .027-.095]). Two patients had an initial troponin measurement that was within normal limits (≤.01 ng/mL) but was subsequently elevated at either 3 or 6 hours. Of these, one reached a maximum value of .03 ng/mL before declining to .01 ng/mL, and the other increased from .01 to .02 ng/mL. A significant delta uptrend in serum cTnT was seen in only 4 patients (1.6%) and these cases are shown in detail in Table 2. All four of these had an initial cTnT level that was elevated. In two of these patients, troponin elevation occurred in the setting of hemodynamic augmentation using intravenous phenylephrine to support cerebral perfusion. In these cases, cTnT elevation was temporally correlated with the initiation