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## Elevated Troponin in Patients with Intracerebral Hemorrhage

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## Elevated Troponin in Patients with Intracerebral Hemorrhage

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### Abstract

Troponin, a known marker for cardiac damage, also plays a role in predicting mortality and morbidity in patients with intracerebral hemorrhage (ICH). Elevated troponin levels have been noted in patients with ICH and studies have shown that such patients are more likely to experience adverse outcomes. The mechanism behind this is suggested to be the release of sympathetic hormones causing myocardial injury. The first differential that often comes to mind when a physician sees an elevated troponin level is a myocardial infarction (MI). In this paper, we present a case of a 54-year-old female who presented to the ED with nausea, vomiting, and severe headache with a troponin level of 47,000. This patient was initially treated with heparin for a non-ST elevation myocardial infarction (NSTEMI), but CT of the head showed ICH. Since part of the treatment protocol for a MI is heparin drip, not recognizing ICH as an underlying cause of MI could lead to worsened hemorrhage and death.

### Keywords

Troponin, Intracerebral Hemorrhage

### Conflict of Interest Statement

We do not have any conflicts of interest to disclose.

### Cover Page Footnote

None

## CASE REPORT

# Elevated Troponin in Patients With Intracerebral Hemorrhage

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## Abstract

Troponin, a known marker for cardiac damage, also plays a role in predicting mortality and morbidity in patients with intracerebral hemorrhage (ICH). Elevated troponin levels have been noted in patients with ICH and studies have shown that such patients are more likely to experience adverse outcomes. The mechanism behind this is suggested to be the release of sympathetic hormones causing myocardial injury. The first differential that often comes to mind when a physician sees an elevated troponin level is a myocardial infarction (MI). In this paper, we present a case of a 54-year-old female who presented to the ED with nausea, vomiting, and severe headache with a troponin level of 47,000. This patient was initially treated with heparin for a non-ST elevation myocardial infarction (NSTEMI), but CT of the head showed ICH. Since part of the treatment protocol for a MI is heparin drip, not recognizing ICH as an underlying cause of MI could lead to worsened hemorrhage and death.

**Keywords:** Troponin, Intracerebral hemorrhage

## 1. Introduction

Intracerebral hemorrhage is the term for bleeding in the brain parenchyma. With an in-hospital mortality rate up to 50%, ICH is considered one of the most dangerous types of strokes.<sup>1</sup> Survivors of stroke often experience disability, including motor deficits, one of the most common functional deficits noticed after stroke. The long-term home care and rehabilitation often required for stroke survivors also places a financial burden on patients and families.<sup>1</sup>

The usage of non-contrast CT to diagnose ICH is well-known, but perhaps a lesser-known phenomenon is the association between ICH and elevated troponin levels.<sup>2</sup> The mechanism proposed to be behind the elevation of troponin in ICH is sympathetic nervous system (SNS) activation leading to the release of catecholamines that can cause myocardial injury.<sup>2</sup> There have been studies that

have shown that patients with ICH who have elevated troponin levels are at higher risk for in-hospital mortality.<sup>3</sup> The neurocardiac mechanism behind elevated troponin levels in ICH is also suspected to lead to ischemia, abnormalities in wall motion, and systolic dysfunction of the left ventricle.<sup>4</sup> The norepinephrine released during sympathetic activation is also surmised to damage nerve terminals.<sup>4</sup> Myocardial damage itself is also associated with decreased cerebral perfusion, which potentially explains why elevated troponin levels in ICH can reflect higher risk of adverse events.<sup>4</sup>

Since elevated troponin is more commonly associated with cardiac damage rather than ICH, this could lead to the deadly mistake of giving heparin to a patient with hemorrhagic stroke. This case presents a 54-year-old female with elevated troponin levels who was eventually found to have an ICH after initially being treated with heparin for a NSTEMI. This report is intended to make more clinicians aware that elevated troponin levels

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combined with the appropriate clinical presentation should prompt further workup for ICH and delay the administration of heparin.

## 2. Case presentation

A 54-year-old female with a past medical history significant for coronary artery disease, chronic kidney disease, hyperlipidemia, hypertension, and diabetes mellitus type 1 presented to the emergency department (ED) due to nausea and vomiting. The patient's symptoms started after having dinner. She vomited 8 times from dinner the previous night up until admission. The vomit was nonbilious and nonbloody. The patient also reported a headache that started the night before admission with a 10/10 intensity and unlike any of the headaches she had in the past. She denied dizziness and confusion. She also denied having shortness of breath and diaphoresis but endorsed chest pain. She took nitroglycerin 3 times for the chest pain and had minimal relief at first but eventually the pain level decreased. She said that the chest pain was intermittent and mild at admission and that she is comfortable despite it. She denied recent travel or sick contacts. Her past surgical history was significant for triple bypass and abdominal aortic aneurysm repair. Her home medications included hydralazine, metoprolol, low dose Aspirin, and nitroglycerin (as needed). Her family history and social history were noncontributory. She denied tobacco and illicit drug usage but reported occasional alcohol usage. Her vitals showed temperature of 98.2 F, heart rate of 86 beats per minute, a blood pressure of 208/117 mmHg, respirations of 18 breathes per minute, and O<sub>2</sub> saturation of 98% on room air. Her physical examination showed a middle-aged female uncomfortable due to severe right temporal headache. Her lungs were clear to auscultation but there were decreased breath sounds at the lower bases bilaterally. The remainder of the physical examination was within normal limits. The labs done in the ED found a troponin level (high sensitivity) of 47,000 pg/mL. Her electrocardiogram showed ST and T wave changes in leads I and aVL (Fig. 1). Her chest x-ray did not show signs of congestive heart failure (CHF). The CT of the abdomen and pelvis

did not show any abnormal findings. The patient was initially thought to have only an NSTEMI. Cardiology was consulted and the patient was placed on the telemetry-monitored floor. The patient was placed on aspirin, labetalol, atorvastatin, and heparin drip. She was also given a loading bolus of heparin drip. Labetalol, morphine, and intravenous (IV) hydralazine were given for blood pressure control. After these orders were placed, non-contrast head CT was performed about an hour after admission since the patient reported having a severe headache that did not improve (Fig. 2). The CT showed right temporal lobe hemorrhage spreading into the ventricles. Neurosurgery was consulted and determined that the patient did not need surgical intervention since she was clinically stable and could be medically managed. Heparin and aspirin were held given the risk of worsening hemorrhage. Protamine was given to reverse the heparin. Beta blocker and statin were continued. Though the patient was initially admitted and treated for NSTEMI only, ICH was added to diagnosis and surmised to be the main cause of the elevated troponin level. The repeat CT showed no significant changes. Troponin trended downwards. Her blood pressure also became more stable. The patient was eventually discharged on Atorvastatin,

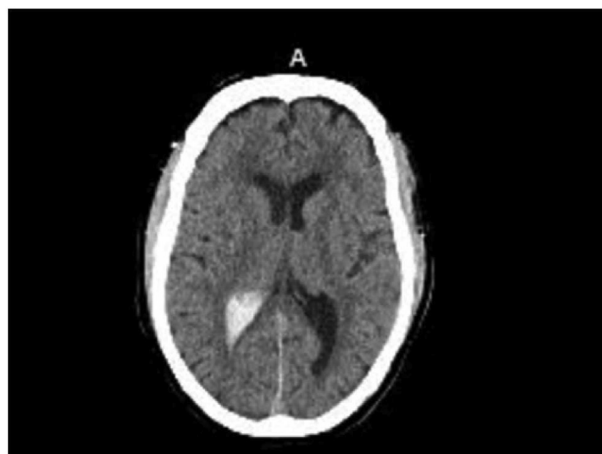


Fig. 2. Head CT showed right temporal lobe hemorrhage spreading into ventricles.



Fig. 1. Electrocardiogram showed ST and T wave changes and QS complexes in leads I and aVL.

Carvedilol, Hydralazine, Isosorbide mononitrate, and Nitroglycerin (prn).

### 3. Discussion

A study done using data from a tertiary medical center in 2013–2015 showed that among 306 patients with ICH, 27% had elevated troponin. The mortality rate in the ICH group was 31.3%.<sup>5</sup> This stresses the importance of remembering that although elevated troponin is widely associated with MI, it can also signify MI secondary to ICH within the proper context. In the case of this patient, she reported both chest pain and headache. The headache was described as severe and unlike any headache she has experienced in the past, warranting a head CT. The American College of Radiology deems that it is usually appropriate to order a head CT without IV contrast if a patient has sudden, severe headache.<sup>6</sup>

When a clinician sees elevated troponin levels, there is often a strong tendency to think mainly from a cardiac perspective. Troponin levels are more often associated with cardiac myocyte damage from myocardial infarction (MI). Although MI should be on one's differential, it is important to consider ICH as well depending on the patient presentation. This patient's type of presentation is the scenario where ICH should be on the differential. As previously mentioned, in this case, the patient reported a severe headache, prompting the clinician to order a CT.

Heparin and aspirin can both be part of the treatment protocol for NSTEMI. In this case, both these medications were initially used in this patient prior to CT results being available. The elevated troponin prompted the clinician to treat her for NSTEMI without considering the repercussions of treating a patient who may have ICH with heparin. As mentioned, she did end up having ICH and the loading bolus of heparin already given may have worsened the hemorrhage in terms of size. Furthermore, heparin is also used in hospitalized patients for thromboembolism prophylaxis. Such usage of anticoagulants and antiplatelet therapy can be deadly in ICH as it can worsen the hemorrhage. It is important to discontinue aspirin and/or heparin if these were started prior to establishing diagnosis of ICH and refrain from using these if ICH is diagnosed beforehand. This leads us to the question when is it acceptable to use anticoagulants and antiplatelet therapy in patients with elevated troponin? The answer is that it depends on the clinical presentation of the patient. NSTEMI and ICH present very differently when viewed individually. The

former often presents with chest pain, while the latter presents with headache. In this case, NSTEMI and ICH presented together, and it may have been appropriate to hold off on anticoagulant/antiplatelet therapy until a head CT result is obtained. On the contrary, if a patient presented with no headache and purely symptoms of NSTEMI, such as chest pain and diaphoresis, then anticoagulant/antiplatelet therapy should be given.

### 4. Conclusion

The recognition that elevated troponin levels can be a result of ICH can help clinicians maintain a high degree of suspicion for ICH. ICH can be missed if a clinician thinks solely from a cardiac standpoint when troponin levels are elevated, and the electrocardiogram shows ischemic changes. Treating a patient with ICH using MI treatment protocol can have dire consequences. To prevent this deadly circumstance, clinicians should be educated more on elevated troponin and ICH and encouraged to keep ICH on their differential when appropriate. Bringing awareness to the predictive value of elevated troponin levels in patients with ICH can assist physicians with clinical decision-making and potentially improve patient outcomes.

### Conflict of interest

We do not have any conflicts of interest to disclose.

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