SVOA Neurology

e-ISSN 2753-9180

ScienceVolks Open Access

Case Report

Troponin T versus Troponin I Acute Myocardial Infarction in a 63 Year Old Female with an Acute Cervical Spinal Cord Injury after Post-Operative Debridement

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https://doi.org/10.58624/SVOANE.2026.07.001

Received: December 17, 2025

Published: January 08, 2026

Citation: Bandeira T. Troponin T versus Troponin I Acute Myocardial Infarction in a 63 Year Old Female with an Acute Cervical Spinal Cord Injury after Post-Operative Debridement. *SVOA Neurology* 2026, 7:1, 01-05. doi. 10.58624/SVOANE.2026.07.001

Abstract

Background: Chronically elevated Troponin T (cTnT) in neurodegenerative and neuromuscular diseases is a recognized but underappreciated phenomenon, often leading to diagnostic challenges. This is particularly critical in spinal cord injury (SCI) patients, where physical examination is limited and atypical presentations of acute coronary syndromes are common. Distinguishing true myocardial infarction from false-positive cTnT elevation is essential to avoid unnecessary interventions.

Case Presentation: A 63-year-old female with chronic T4 paraparesis presented with an acute cervical SCI. Following a surgical debridement, she developed acute dyspnea, hypotension, bradyarrhythmia, and hypoxia. Bedside echocardiogram revealed antero- and inferoseptal hypokinesia. Serial biomarkers showed a >20% rise in both cTnT (96.4 to 264 pg/mL) and Troponin I (cTnI) (0.110 to 0.610 µg/L), meeting criteria for non-ST-elevation myocardial infarction (NSTEMI). However, while cTnI normalized within days, cTnT remained persistently elevated (42.1 pg/mL) one month post-event, against a backdrop of chronic muscle atrophy and severe pressure ulcers.

Conclusion: This case highlights the diagnostic utility of cTnI in the SCI population. In patients with chronic neuromuscular degeneration, cTnI demonstrates the expected rise-and-fall dynamic of an acute myocardial injury, whereas cTnT exhibits chronic elevation likely due to skeletal muscle pathology. For the differential diagnosis of NSTEMI in SCI patients, cTnI appears to be a more reliable biomarker, potentially preventing misinterpretation and unnecessary cardiac interventions.

Keywords: *Spinal Cord Injury, Troponin I (cTnI), Troponin T (cTnT), Non-ST Elevation Myocardial Infarction (NSTEMI), Neurodegenerative Diseases, Paraplegia / Paraparesis, diagnostic Error*

Introduction

Chronically elevated Troponin T is a recognized, yet under-discussed and insufficiently explored phenomenon in neurodegenerative diseases. For example, skeletal myopathies are frequently associated with non-cardiac Troponin T elevation [5]. This lack of data complicates the interpretation of clinical scenarios, leading physicians not only to errors in clinical judgment but also to unnecessary supplementary testing and interventions [5,6]. This diagnostic challenge is particularly acute in spinal cord injury (SCI) centres. Here, the physical examination is often difficult, and the interpretation of findings is complicated by frequently reduced or absent sensation, which varies according to the neurological level of injury.

Consequently, a suspicion of NSTEMI can be misleading due to false-positive Troponin T elevation. Conversely, in cases of atypical NSTEMI presentation, a myocardial infarction may go unnoticed. Just as often, a clinical picture may be misinterpreted as NSTEMI, frequently leading to unnecessary interventions such as angioplasty.

There is little reference in the literature to the dynamics of cardiac Troponin T (cTnT) and I (cTnI) in neuromuscular and neurodegenerative diseases, and practically none addressing cTnT and cTnI variations in acute and chronic spinal cord injury patients [5, 6, 7, 10].

Case Presentation

A 63-year-old female with obesity was admitted following an acute cervical spinal cord injury (SCI) secondary to known C3-4 spinal stenosis. The patient had a pre-existing chronic T4 paraparesis. Her medical history was complicated by type 2 Diabetes Mellitus with multiple leg ulcers, a right foot amputation, and a subsequent distal metatarsal amputation. In addition to her wound healing disorder and SCI, she had a longstanding stage IV sacral pressure ulcer. One week after undergoing cervical spine surgery, a debridement of this pressure ulcer was performed under general anesthesia.

The patient remained in the recovery room for 30-45 minutes without major complications before returning to her medical ward. Upon arrival to the ward, she developed significant dyspnea with an oxygen saturation of 80%, hypotension (80/40 mmHg), bradyarrhythmia (48 beats per minute), and a diminished level of consciousness. She was immediately administered three liters of oxygen, a 1000ml Ringer's infusion, and 20 drops of Etilefrine Hydrochloride. Following this, her vital parameters normalized and she spontaneously regained a regular heart rhythm.

However, she reported persistent subjective dyspnea, despite maintaining 98% oxygen saturation on 1 liter of oxygen.

Investigations

The ECG revealed nonspecific pathological R-wave progression in the precordial leads and a first-degree AV block. A bedside echocardiogram detected hypokinesis of the anteroseptal and inferoseptal walls. Troponin levels showed a significant rise within three hours: Troponin T increased from 96.4 pg/mL to 264 pg/mL, and Troponin I rose from 0.110 µg/L to 0.610 µg/L. Against the background of a probable myocardial infarction, the patient was transferred to the intensive care unit for further management.

An invasive coronary intervention was ultimately not performed as the patient showed progressive stabilization. Subsequent laboratory analysis revealed a key divergence: Troponin T remained above normal values in all later measurements (peak 264 pg/mL, lowest 42.1 pg/mL one month later), while Troponin I paralleled a normal decline after the event (peak 0.610 µg/L, lowest 0.010 µg/L).

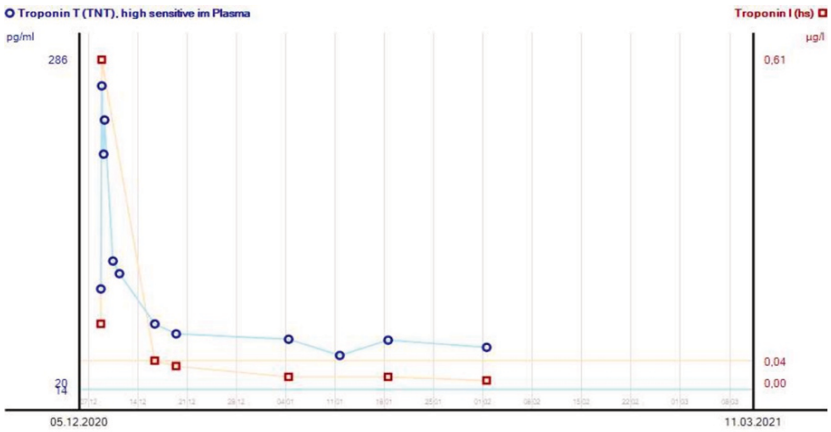

Figure 1. Troponin T versus Troponin I with chronic increased TnT Values.

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Differential Diagnosis

Myocardial infarction (MI) is defined as a clinical event in the setting of myocardial ischemia with evidence of myocardial injury [1,2]. The diagnosis is confirmed by a rise and/or fall in cardiac troponin (T or I) values, supported by typical symptoms, suggestive ECG changes, or imaging evidence of new regional wall motion abnormality [1,2].

The persistent elevation of cTnT and CK-MB in the absence of a corresponding cTnI elevation or typical symptoms is notable. This pattern is likely attributable to the fact that cTnI is not expressed in skeletal muscle at any stage of development or regeneration, whereas cTnT isoforms can be re-expressed in regenerating skeletal myofibers following injury [5, 6, 7, 9]. This phenomenon has been described in asymptomatic patients with neurological diseases but without cardiac injury. Therefore, while cTnI is a structural component of cardiac muscle and would be expected to follow the acute rise-and-fall pattern of an MI, cTnT can be chronically elevated from a skeletal muscle source. Given that this patient later presented without typical MI symptoms and has a chronic SCI, cTnI emerges as a critical tool in the differential diagnosis to help exclude a false-positive MI result.

Discussion

The diagnostic evaluation of acute myocardial infarction in patients with complex chronic neuromuscular conditions, such as spinal cord injury (SCI), represents a significant clinical challenge. This case vividly illustrates the peril and promise of modern cardiac biomarkers in this setting. It reinforces a critical axiom: in medicine, the test result is not the diagnosis; the diagnosis is the interpretation of the test result within the full clinical context [1, 2].

Our patient's presentation was classically ambiguous. She possessed multiple potent triggers for myocardial demand ischemia: the physiological stress of two major surgeries in one week, potential peri-operative volume shifts, probable autonomic dysreflexia, and significant underlying coronary artery disease risk from diabetes and chronic inflammation [3]. The concomitant findings of acute dyspnea, regional wall motion abnormality on echocardiography, and a significant serial rise in both cTnI and cTnT initially satisfied the Fourth Universal Definition of Myocardial Infarction [1].This justified the acute management for NSTEMI.

However, the subsequent discordant biomarker kinetics revealed a more complex pathophysiology. The rapid rise-and-fall of cTnI is the archetypal pattern of acute myocardial necrosis [4]. Its normalization within days confirmed a time-limited cardiac injury. In stark contrast, the persistently elevated cTnT—remaining pathologically high one month post-event— signals a separate, ongoing process. This dissociation is the crux of the diagnostic insight and forms the basis for a critical clinical recommendation.

The explanation could lie in the fundamental biology of the troponin isoforms. Cardiac Troponin I is encoded by a unique gene (*TNNI3*) and its expression is highly restricted to mature cardiomyocytes. No credible evidence exists for its expression in healthy or regenerating skeletal muscle at any stage of development or disease [5, 6]. Therefore, serum cTnI is a highly specific, though not perfectly sensitive, footprint of cardiac myocyte damage.

Cardiac Troponin T, encoded by *TNNT2*, presents a different story. While the current high- sensitivity assays are designed for cardiac-specific epitopes, it is well-established that isoforms of Troponin T are re-expressed in regenerating skeletal myofibers following injury or in chronic disease states [5, 7, 8]. This phenomenon has been documented in Duchenne muscular dystrophy, polymyositis, and critically ill patients with rhabdomyolysis [6, 7, 9]. Our patient presented a textbook scenario for sustained skeletal muscle turnover: chronic denervation atrophy from her complete T4 paraplegia, acute superimposed denervation from the cervical injury, and a profound catabolic state driven by severe stage IV pressure ulcers and infected diabetic wounds. This creates a persistent, low-grade "leak" of cTnT from skeletal muscle, establishing an elevated baseline. The acute physiologic insult likely caused a superimposition of true cardiac release upon this elevated baseline, resulting in the dramatic initial spike.

- **Primary Reliance on cTnI:** For the acute assessment of suspected ACS in any patient with known or suspected chronic neuromuscular disease (SCI, muscular dystrophy, advanced ALS), cTnI should be the primary biomarker guiding management. Its kinetic profile (delta change) provides a clear signal for acute cardiac injury against a typically silent background.

- **Cautious Interpretation of cTnT:** An elevated cTnT level must be interpreted with extreme caution. A single value is virtually uninterpretable without a prior baseline. In the acute setting, the **dynamic change (delta)** of cTnT is more informative than its absolute value. A chronic, stable elevation is more likely reflective of skeletal myopathy than active coronary disease.

- **The Pitfall of False-Positive Leads to Harm:** Misinterpreting a chronically elevated cTnT as an acute MI can trigger a cascade of low-value, high-risk care: unnecessary ICU transfer, prolonged hospital stay, invasive coronary angiography with its associated risks of contrast nephropathy and vascular injury, and potentially unwarranted coronary intervention [10]. These interventions are not benign, especially in a fragile population with SCI.

- **The Risk of False-Negative:** Conversely, assuming all troponin elevation is "chronic" poses the opposite danger. An atypical MI (e.g., silent ischemia due to autonomic dysfunction) could be missed if clinicians dismiss a genuine acute rise in *both* biomarkers, particularly cTnI.

Limitations and Future Directions

Our report has limitations. This is a single case, and we lacked a pre-injury baseline troponin level for this patient, which would have been invaluable. Furthermore, while we strongly implicate skeletal muscle as the source of chronic cTnT, we cannot entirely rule out other chronic cardiac pathologies (e.g., low-grade myocarditis, microvascular dysfunction) associated with her comorbidities.

This case highlights a glaring gap in the literature and clinical practice. Future research should aim to:

1. Establish the prevalence and magnitude of chronic cTnT elevation in a cohort of SCI patients, stratified by level, completeness, and duration of injury.

2. Prospectively evaluate the diagnostic performance of cTnI versus cTnT in SCI patients presenting with suspected ACS.

3. Develop evidence-based guidelines or decision pathways for emergency and rehabilitation physicians facing this common dilemma.

Conclusion

In conclusion, this case report serves as a critical exemplar in neuro-cardiology. It moves beyond merely describing a biochemical curiosity to offering a practical framework for clinical decision-making. In patients with spinal cord injury, "troponin" is not a single entity. A dual- marker strategy, recognizing cTnI as the specific arbiter of acute injury and cTnT as a potential confounder from skeletal muscle, is essential. By integrating biomarker-specific kinetics with the clinical context, clinicians can avoid diagnostic errors, optimize resource utilization, and provide safer, more precise care for this vulnerable patient population.

Learning points/ Take home message

1. Differential Biomarker Dynamics are Key: In patients with spinal cord injury or chronic neuromuscular disease, Troponin I (cTnI) and Troponin T (cTnT) can exhibit divergent long-term patterns. A concurrent rise in both may indicate acute myocardial injury, but a persistent, isolated elevation of cTnT is highly suggestive of a non-cardiac, skeletal muscle origin.

2. cTnI as the Preferred Biomarker in SCI: For evaluating suspected acute coronary syndromes in SCI patients, cTnI is the more reliable and clinically actionable biomarker. Its specificity for cardiac tissue and lack of expression in regenerating skeletal muscle make its dynamic (rise and fall) a trustworthy indicator of true myocardial necrosis.

3. Avoid Diagnostic Pitfalls: Physicians must be aware that chronic cTnT elevation is a common finding in this population due to generalized muscle atrophy, pressure ulcers, and underlying neurodegenerative processes. Interpreting such elevation in isolation can lead to overdiagnosis of NSTEMI, resulting in unnecessary transfers, testing, and potentially invasive interventions like angiography.

4. Context-Dependent Interpretation: The diagnosis of MI in SCI patients requires integrating biomarker trends (preferentially cTnI) with clinical context and imaging findings, as typical symptoms may be absent or masked. A holistic view is essential to distinguish between a true cardiac event and a chronic, non-cardiac troponin elevation.

Conflict of Interest

The author declare no conflict of interest.

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