Çukurova Tıp Öğrenci Dergisi
ÇUKUROVA ÜNİVERSİTESİ TIP FAKÜLTESİ

Çukurova Tıp Öğrenci Derg. 2026;6(1):46-51

OLGU SUNUMU / CASE REPORT

Markedly elevated high-sensitivity troponin I in severe polytrauma without acute coronary syndrome: case report

Çoklu travma hastasında akut koroner sendrom olmadan saptanan troponin I yüksekliği: vaka sunumu

Broboană Anişoara Elena[1], Ömer Taşkın[2]

[1]University of Medicine and Pharmacy of Craiova, Craiova, Romania
[2]Cukurova University, Adana, Türkiye

Abstract

Interpreting markedly elevated cardiac biomarkers in severe polytrauma presents a recurrent diagnostic dilemma, requiring the differentiation of acute coronary syndrome from trauma-related injury and rhabdomyolysis. A 54-year-old woman with no known comorbidities was admitted to the emergency department following an accidental fall from approximately 5 meters. Initial evaluation revealed hypotension requiring fluid resuscitation. Contrast-enhanced computed tomography demonstrated multiple injuries, including right-sided pneumothorax, rib fractures, pelvic fractures classified as Young-Burgess lateral compression type I, and a grade II renal laceration. The electrocardiogram showed no signs of acute ischemia at any time point. High-sensitivity troponin I peaked at 1097.8 nanograms per liter (approximately 27 times the upper reference limit), while creatine kinase and creatine kinase-MB continued to rise, a pattern incompatible with acute myocardial infarction and consistent with concomitant rhabdomyolysis. Transthoracic echocardiography was normal. Acute coronary syndrome was excluded by cardiology evaluation, and invasive coronary investigation was not pursued. This case highlights the importance of integrating clinical context, electrocardiographic findings, echocardiography, and biomarker kinetics when interpreting markedly elevated cardiac biomarkers in polytrauma patients.

Keywords: Multiple trauma, thoracic injuries, troponin I, rhabdomyolysis, acute coronary syndrome, emergency service

Öz

Çoklu travma hastalarında belirgin şekilde yükselen kardiyak biyobelirteçlerin yorumlanması; akut koroner sendromun travmaya bağlı hasar ve rabdomiyolizden ayırt edilmesini gerektiren, sıklıkla karşılaşılan bir tanısal ikilemdir. Bilinen herhangi bir ek hastalığı olmayan 54 yaşındaki bir kadın, yaklaşık 5 metre yükseklikten kaza sonucu düşme sonrasında acil servise getirildi. İlk değerlendirmede, sıvı resüsitasyonu gerektiren hipotansiyonu mevcuttu. Yapılan kontrastlı bilgisayarlı tomografi incelemesinde sağda pnömotoraks, kot kırıkları, Young-Burgess lateral kompresyon tip I pelvis kırığı ve evre II böbrek laserasyonu dahil olmak üzere çoklu yaralanmalar tespit edildi. Başvuru anında ve sonrasında çekilen elektrokardiyografilerin hiçbirinde akut iskemik bulguya rastlanmadı. Yüksek duyarlıklı troponin I seviyesi 1097,8 ng/L'ye (üst referans sınırının yaklaşık 27 katı) ulaşırken; kreatin kinaz ve kreatin kinaz-MB seviyeleri sürekli artış gösterdi. Bu patern, akut miyokard enfarktüsü ile uyumsuz olup eşzamanlı rabdomiyoliz ile uyumluydu. Transtorasik ekokardiyografi normal olarak değerlendirildi. Kardiyoloji değerlendirmesi sonucunda akut koroner sendrom dışlandı ve invaziv koroner girişime başvurulmadı. Bu vaka, çoklu travma hastalarında belirgin şekilde yükselmiş kardiyak biyobelirteçleri yorumlarken klinik bağlam, elektrokardiyografik bulgular, ekokardiyografi ve biyobelirteç kinetiklerini bir araya getirmenin önemini vurgulamaktadır.

Anahtar kelimeler: Çoklu travma, toraks yaralanması, troponin I, rabdomiyoliz, akut koroner sendrom, acil servis

Yazışma Adresi/Address for Correspondence: Broboană Anişoara Elena, University of Medicine and Pharmacy of Craiova, Romania, (Erasmus mobility at Çukurova University Faculty of Medicine, Adana, Türkiyey)
E-mail: anisoarabroboana13@gmail.com
Geliş tarihi/Received: 30.04.2026 Kabul tarihi/Accepted: 08.05.2026

INTRODUCTION

Falls from height are a frequent mechanism of high-energy blunt trauma commonly associated with multisystem injuries. Blunt thoracic trauma accounts for nearly one third of severe trauma cases, and blunt cardiac injury (BCI) has been reported in 3% to 56% of these patients depending on diagnostic criteria[1,2]. BCI encompasses a broad spectrum, from clinically silent myocardial contusion to lethal arrhythmias and cardiac rupture.

Current Eastern Association for the Surgery of Trauma (EAST) guidelines recommend combined electrocardiography (ECG) and troponin I measurement for BCI screening; both must be normal to exclude clinically significant injury[3]. High-sensitivity troponin I (hs-cTnI) assays have improved sensitivity but reduced specificity, with troponin elevation reported in up to 44% of blunt thoracic trauma patients[4], reflecting myocardial contusion, hypoperfusion, catecholamine-mediated stress, or systemic inflammation.

Interpreting markedly elevated cardiac biomarkers in polytrauma is a recurrent diagnostic dilemma. The clinician must distinguish acute coronary syndrome (ACS) from trauma-related myocardial injury, while accounting for the limited specificity of creatine kinase-MB (CK-MB) in the presence of rhabdomyolysis[5]. In this case report we present a severe polytrauma patient with markedly elevated hs-cTnI (peak 1097.8 ng/L, approximately 27 times the upper reference limit) and progressively rising CK-MB, in whom ACS was excluded based on integrated clinical, electrocardiographic, echocardiographic, and biomarker assessment.

CASE

A 54-year-old woman with no comorbidities was presented to a peripheral hospital following an accidental fall from approximately 5 meters. Initial evaluation revealed hypotension (60/40 mmHg), heart rate 80 bpm, Glasgow Coma Scale 15, and oxygen saturation 98%. Intravenous resuscitation with 1000 mL of 0.9% saline was initiated, and the patient was transferred to our tertiary care center. Written informed consent was obtained from the patient for publication of this case report and accompanying images.

On arrival, vital signs had stabilized (blood pressure 104/73 mmHg, heart rate 70 bpm, respiratory rate 20/min, oxygen saturation 100%). Physical examination revealed diminished breath sounds in the right hemithorax; cardiac auscultation was normal without pericardial friction rub. Focused Assessment with Sonography for Trauma (FAST) demonstrated free fluid in the right suprarenal area. Contrast-enhanced whole-body computed tomography, without active extravasation, revealed: a 4 cm right-sided pneumothorax with bilateral pleural effusions; fractures of the right 2nd, 3rd, and 6th ribs, right clavicle, and right scapula; a fracture of the L5 spinous process; a grade II (AAST) laceration of the right kidney with perirenal fluid; and pelvic fractures classified as Young-Burgess lateral compression type I (LC-1), with free pelvic fluid. A right thoracostomy tube was placed.

The first 12-lead ECG, showed sinus rhythm at 67 beats per minute (bpm) with left axis deviation, normal intervals (PR 111 ms, QRS 87 ms, QTc 465 ms), and an isolated supranodal extrasystole (Figure 1). There were no ST-segment changes, no pathological Q waves, and no conduction abnormalities suggestive of acute ischemia. A follow-up ECG performed approximately 14 hours later showed sinus tachycardia at 90 bpm and no new ischemic findings.

Laboratory findings are summarized in Table 1. Notably, hs-cTnI was already elevated on presentation (4.5 hours after the accident, 859.1 ng/L), peaked at 1097.8 ng/L at three hours, and declined to 443 ng/L by day 1 morning. In contrast, creatine kinase (CK) rose progressively (1420 to 2295 U/L), and CK-MB increased from 35.7 to 71.5 ng/mL. Marked leukocytosis (22.4 × 10^3/μL), mild hypocalcemia (7.92 mg/dL), and hyperglycemia (175 mg/dL on day 1) were present (Figure 2). D-dimer was markedly elevated (31.741 μg/mL) with normal coagulation parameters. Two units of packed red blood cells were transfused, and tranexamic acid was administered.

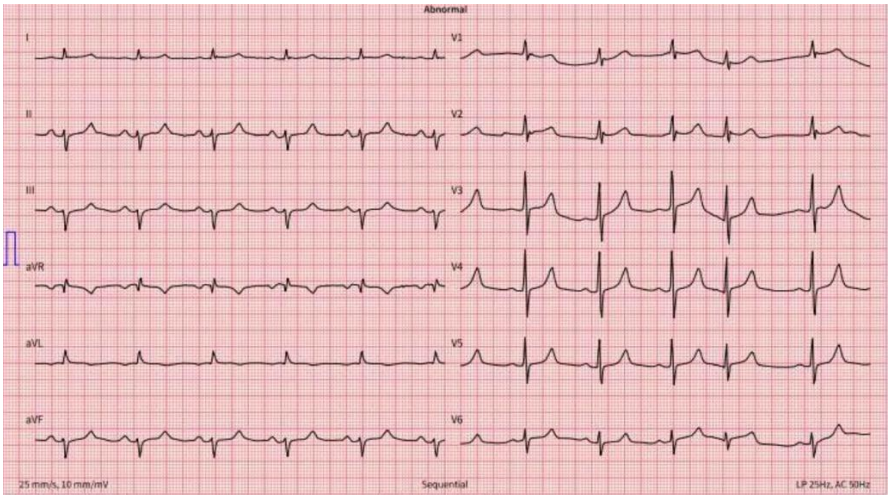

Figure 1. Twelve-lead electrocardiogram on admission. Sinus rhythm at 67 bpm with left axis deviation (-52°), PR 111 ms, QRS 87 ms, QTc 465 ms. An isolated supranodal extrasystole is noted. No ST-segment changes, pathological Q waves, or conduction abnormalities are present. No electrocardiographic criteria for acute coronary syndrome.

Multidisciplinary evaluation included cardiology, which excluded ACS based on the absence of ischemic symptoms, absence of dynamic ECG changes, and the traumatic mechanism. Coronary angiography was not performed. Transthoracic echocardiography on day 1 demonstrated normal cardiac structure and function with physiological pericardial fluid and no tamponade. Low-molecular-weight heparin was prescribed for thromboprophylaxis. The patient was admitted to the intensive care unit. At the time of manuscript preparation (day 7), she remained in stable condition; definitive orthopedic management and long-term follow-up were pending.

Table 1. Temporal evolution of laboratory parameters during the first 28 hours of admission.

Parameter (units)	Reference range	Presentation (~16:54)	19:47	Day 1 (~05:23)	Day 1 (~21:17)
Hemoglobin (g/dL)	12.0-16.0	10.1	8.4	10.6*	9.2
RBC ($\times 10^6/\mu L$)	4.0-5.2	3.36	2.81	3.64	3.16
WBC ($\times 10^3/\mu L$)	4.0-10.0	22.4	18.72	14.58	12.3
AST (U/L)	<35	205	143	121	91
ALT (U/L)	<35	156	122	111	90
hs-cTnI (ng/L)	<40	859.1	1097.8	443	—
CK (U/L)	<234	—	1420	2295	—
CK-MB (ng/mL)	<4.94	35.7	—	71.5	—
INR	0.8-1.2	1.08	—	0.98	0.93
aPTT (s)	26-40	26.2	—	24.7	23.5
D-dimer (µg/mL)	0-5	—	—	31.741	—
Sodium (mmol/L)	136-144	143	—	141	140
Potassium (mmol/L)	3.6-5.1	—	—	5.29	4.41
Calcium (mg/dL)	8.4-9.7	7.92	—	8.3	7.9
Magnesium (mg/dL)	1.8-2.5	1.88	—	1.78	1.72
Glucose (mg/dL)	70-100	101	—	175	114
eGFR (mL/min/1.73m²)	>90	—	—	—	99

*Following transfusion of 2 units of packed red blood cells. RBC: red blood cells; WBC: white blood cells; AST: aspartate aminotransferase; ALT: alanine aminotransferase; hs-cTnI: high-sensitivity troponin I; CK: creatine kinase; CK-MB: creatine kinase-MB; INR: international normalized ratio; aPTT: activated partial thromboplastin time; eGFR: estimated glomerular filtration rate.

Table 2. Summary of traumatic injuries identified on imaging.

Anatomical region	Injury	Imaging
Head and neck	No cranial fractures; no parenchymal hemorrhage or edema	CT
Cervical spine	No fractures	CT
Thorax	Right pneumothorax (4 cm); bilateral pleural effusions; fractures of right ribs 2, 3, 6; right clavicle and scapula fractures	CT, CXR
Abdomen	Right renal laceration (15 × 5 mm, AAST grade II); bilateral perirenal fluid; no pneumoperitoneum	CT, FAST
Pelvis	Sacral, pelvic ring, and right acetabular fractures (Young-Burgess LC-1); free pelvic fluid	CT, PXR
Spine	Fracture of L5 spinous process	CT

CT: computed tomography; CXR: chest radiograph; FAST: Focused Assessment with Sonography for Trauma; PXR: pelvic radiograph; AAST: American Association for the Surgery of Trauma.

DISCUSSION

This case illustrates the diagnostic challenge of interpreting markedly elevated cardiac biomarkers in polytrauma. Our patient presented with hs-cTnI peaking at 27 times the upper reference limit, accompanied by rising CK and CK-MB, without ischemic symptoms, ECG evidence of ACS, or echocardiographic abnormalities.

Troponin elevation in blunt thoracic trauma is multifactorial: direct myocardial contusion (particularly of the anteriorly positioned right ventricle), supply-demand mismatch from hypoperfusion, catecholamine-mediated injury and systemic inflammation may all contribute[1,6]. In our patient initial hypotension, blood loss requiring transfusion, and high-energy thoracic impact with multiple rib fractures plausibly accounted for troponin release without implying clinically significant BCI. Recent prospective data confirm that up to 73% of polytrauma patients develop troponin elevation within 24 hours, frequently through delayed mechanisms[2].

The most instructive feature is the discordant biomarker kinetics. Hs-cTnI rose from 859.1 ng/L to a peak of 1097.8 ng/L at three hours, then declined rapidly to 443 ng/L, while CK rose from 1420 to 2295 U/L and CK-MB from 35.7 to 71.5 ng/mL (Figure 2). This pattern is incompatible with acute myocardial infarction, in which CK-MB peaks at 18-24 hours and troponin plateaus over 24-72 hours[7]. CK-MB, although enriched in cardiac muscle (25-30% of total cardiac CK activity), is also expressed in skeletal muscle at 1-20% depending on the muscle group[8]. In the context of massive rhabdomyolysis evidenced by markedly elevated CK, transaminases, lactate dehydrogenase, and developing hyperkalemia (5.29 mmol/L) from myocyte lysis, CK-MB elevation cannot be attributed to myocardial origin. The EAST guideline explicitly recommends against CK isoenzyme analysis for BCI screening, as it does not predict complications[3]. Our case supports this recommendation: CK-MB continued to rise due to extensive skeletal muscle injury, while hs-cTnI was already declining, demonstrating that CK-MB elevation in this setting reflected rhabdomyolysis rather than ongoing myocardial damage.

Several elements supported ACS exclusion. The patient had no cardiovascular risk factors, no ischemic symptoms and no electrocardiographic criteria for myocardial infarction at any time point. Electrocardiographically, both admission and follow-up tracings were within normal limits apart from an isolated supranodal extrasystole, with no ST-segment changes, no pathological Q waves, no conduction abnormalities and no dynamic evolution suggestive of ischemia. The borderline QTc (465 ms) normalized to 454 ms on the follow-up tracing and was plausibly contributed to by mild hypocalcemia and hypomagnesemia during resuscitation with citrate-containing blood products. Echocardiography excluded structural cardiac abnormality and tamponade. The rapidly declining hs-cTnI, markedly elevated D-dimer (consistent with compensated trauma-induced coagulopathy), and the coherent traumatic mechanism provided no reasonable pretest probability for coronary intervention. Proceeding to angiography would have exposed an actively bleeding patient to contrast load, dual antiplatelet therapy and procedural hemorrhagic risk without expected benefit.

Limitations include the absence of cardiac magnetic resonance imaging and incomplete follow-up at the time of writing. Despite these, the case illustrates how integrated assessment, combining mechanism, clinical presentation, the absence of ischemic electrocardiographic changes, and biomarker kinetics allows safe ACS exclusion in polytrauma. In the era of high-sensitivity troponin assays, clinicians should remain aware that very high troponin values do not mandate invasive evaluation when the clinical context is incompatible with a coronary event.

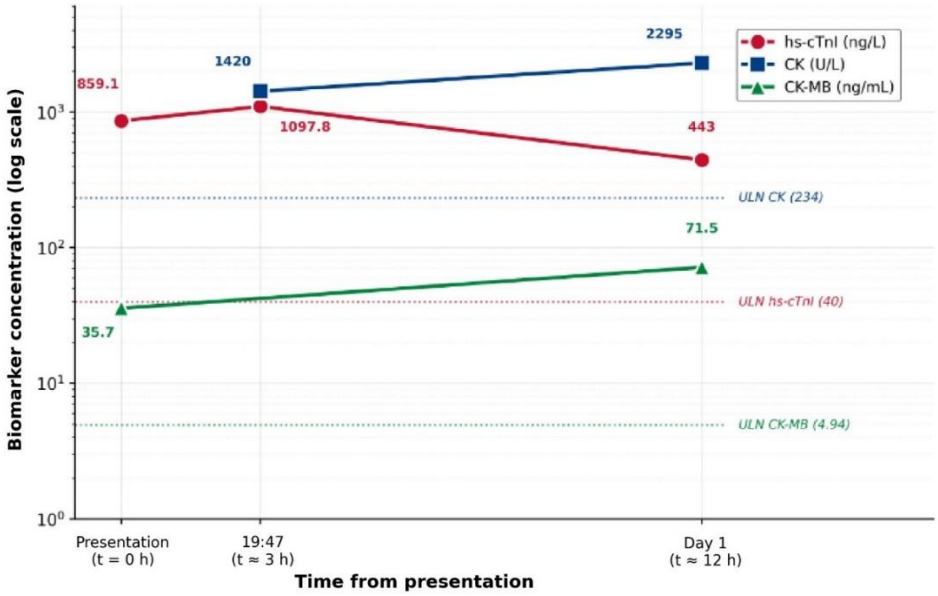

Figure 2. Temporal evolution of cardiac biomarkers during the first 12 hours after admission. Hs-cTnI peaked at 1097.8 ng/L and declined rapidly to 443 ng/L by the following morning, while CK and CK-MB continued to rise. This discordant pattern is incompatible with a typical acute myocardial infarction time course and is consistent with concomitant rhabdomyolysis from extensive musculoskeletal injury. Dotted horizontal lines indicate upper reference limits.

In conclusion, markedly elevated cardiac biomarkers in polytrauma patients do not, in themselves, mandate investigation for ACS. In our patient, integrated assessment of mechanism, clinical presentation, an ECG without ischemic changes, and the discordant kinetics of hs-cTnI and CK-MB allowed safe exclusion of acute coronary syndrome and avoided invasive coronary angiography in the setting of active hemorrhage. Clinicians should remain particularly cautious when interpreting CK-MB in the presence of extensive musculoskeletal injury, as concomitant rhabdomyolysis substantially limits its specificity for myocardial origin. A structured approach combining ECG, hs-cTnI trend, and clinical context remains the cornerstone of cardiac assessment in blunt polytrauma.

Author Contributions: Concept/Design : BAE; Data acquisition: BAE, ÖT; Data analysis and interpretation: BAE; Drafting manuscript: BAE; Critical revision of manuscript: BAE, ÖT; Final approval and accountability: BAE, ÖT; Technical or material support: -; Supervision: ÖT; Securing funding (if available): n/a.
Ethical Approval: This study does not require Ethics Committee Permission/Approval.
Written informed consent was obtained from the patient for publication of this case report and accompanying images.
Peer-review: Externally peer-reviewed.
Conflict of Interest: Authors declared no conflict of interest.
Financial Disclosure: Authors declared no financial support

REFERENCES

1. Biffl WL, Fawley JA, Mohan RC. Diagnosis and management of blunt cardiac injury: what you need to know. J Trauma Acute Care Surg. 2024;96:685-93.
2. Sztulman L, Pfeiffer V, Saenger M, Brenner R, Usov L, Marzi I et al. Beyond the initial impact: troponin

patterns frequently reveal delayed cardiac injury in polytrauma patients. World J Emerg Surg. 2026;21:10.
3. Clancy K, Velopulos C, Bilaniuk JW, Collier B, Crowley W, Kurek S et al. Screening for blunt cardiac injury: an Eastern Association for the Surgery of Trauma practice management guideline. J Trauma Acute Care Surg. 2012;73:301-6.
4. Singh S, Heard M, Pester JM, Angus LD. Blunt cardiac injury. In: StatPearls. Treasure Island, StatPearls Publishing. 2024.
5. Becker ER, Price AD, Whitrock JN, Smith M, Baucom MR, Makley AT et al. Re-evaluating the use of high sensitivity troponin to diagnose blunt cardiac injury. J Surg Res. 2024;300:150-6.
6. Lippi G, Lavie CJ, Sanchis-Gomar F. Detecting cardiac injury: the next generation of high-sensitivity cardiac troponins improving diagnostic outcomes. Clin Chem Lab Med. 2025;63:1941-51.
7. Aujla RS, Patel R. Creatine kinase MB: diagnostic utility and limitations. In: StatPearls. Treasure Island, FL, StatPearls Publishing, 2026.