University Heart Journal
Vol. 21, No. 1, January 2025

CASE REPORT

Acute Coronary Syndrome in the Context of Normal Cardiac Troponin Level- A Case Report

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Abstract:

Acute coronary syndrome (ACS), counted globally as the leading cause of mortality in recent times. Typical clinical presentation of ACS is due to a mismatch between the supply and demand of oxygen (O2) which leads to cardiac ischemia and necrosis. Troponin I, A serum biomarker of myocardial injury has a 99% sensitivity rate in identifying the myocardial damage, and very few instances may deviate from this norm. We report a rare case of ACS where repeated testing of troponin levels returned negative results, even after tests conducted at three different laboratories.

University Heart Journal 2025; 21(1): 53-57
DOI: https://doi.org/10.3329/uhj.v21i1.84437

Introduction:

ACS is the spectrum of clinical presentations caused by acute ischemia of the myocardium due to disruption in the blood flow through coronary arteries; includes ST-elevation myocardial infarction (STEMI) AND non-ST elevation ACS. Acute coronary syndrome may manifest as a STEMI when a thrombus totally obstructs an epicardial coronary artery (Mokhtari, A. et al., 2016). The diagnosis of STEMI relies on clinical features with ST segment elevation in electrocardiography (ECG) (Kumar, A. and Cannon, C.P., 2009). Those patients requiring urgent coronary intervention. Troponin levels may be normal at the time of initial presentation (McNeil, A., 2007).

We present a rare and exceptional case of a 50-year-old man who experienced acute coronary syndrome despite consistently normal cardiac damage markers.

Case Report:

Mr. X, 50 years of age, non-smoker and non-alcoholic, is a businessman with a known history of type II diabetes for 2 years, and hypertension for the same duration. He is a chronic Hepatitis B virus carrier. He was admitted to the BSMMU emergency department with complaints of sudden onset of central chest pain for last 24 hours. The pain commenced when he was seated in a chair at home. It was diffuse and characterized as crushing, with severe intensity and he rated the pain 9 out of 10. The discomfort was accompanied by profuse sweating and several episodes of vomiting. He promptly proceeded to a renowned hospital to seek consultation with a physician. Some baseline study was recommended with an ECG which reveals Acute STEMI with RV infarction; the patient was advised to undergo emergency hospitalization. However, the patient declined, and after one day, was admitted to the BSMMU emergency department for further management.

During Admission in BSMMU emergency he was conscious and oriented, having mild to moderate chest tightness which he rated as 6 out of 10.Regarding his vital, Blood Pressure- 140/90mmHg, pulse-96b/min, temp- 98F, SPO2- 94% with 2L O2 via Nasal cannula. He is not anemic, nonicteric and other systemic examination reveals no abnormalities. We diagnosed the patient as a case of Acute STEMI (Inferior) with RV infarction-delayed arrival. We planned for CAG as patient was having ongoing chest pain.

Echocardiography revealed Basal and midsegment of Inferior wall is hypo-kinetic.There is hypertrophy of IVS and mild LV systolic dysfunction with LVEF 52%

The coronary angiography (CAG) revealed that,

- The left main coronary artery is normal, disease-free with good caliber.

Received: 5 December, 2024 **Accepted:** 17 December, 2024

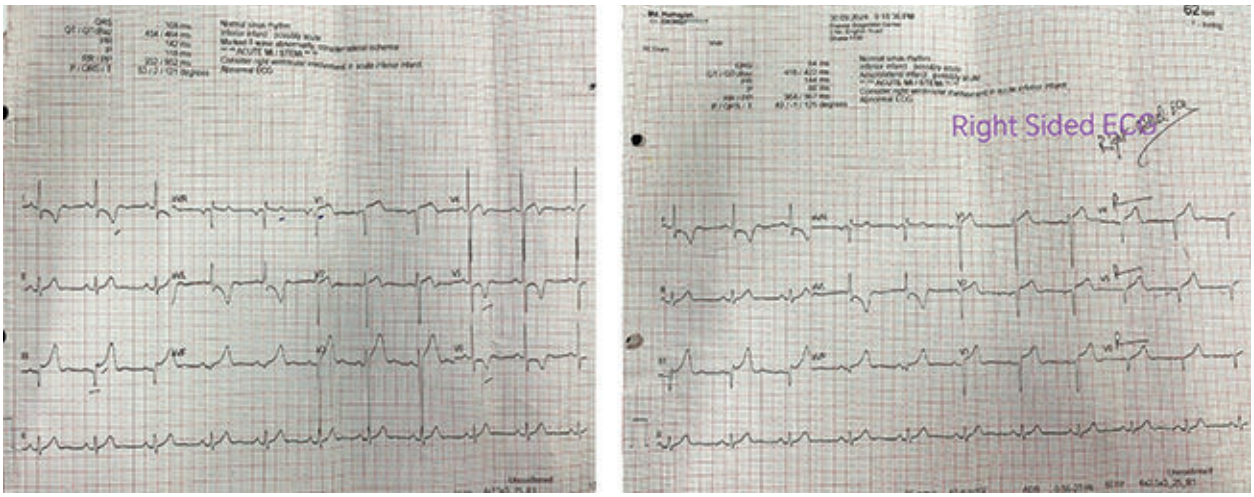

Figure 1: *Initial ECG shows ST segment elevation in lead II, III and aVF & V1 with receprocal ST Segment depression in lead I & aVL. Right sided ECG shows ST elevation in V4R, V5R and V6R.*

- Right coronary artery is the Co-dominant vessel with 90-95% proximal stenosis with G-I thrombus burden
- Left Circumflex Artery exhibits 90% stenosis in mid part.
- The LAD, A Type 4 vessel with minimal plaque and Diagonal-1 displays 99% osteoproximal stenosis.

We used everolimus-eluting stents for angioplasty of the LCx and RCA. The first stent measures 2.5x18mm in the right coronary artery (RCA) and 2.5x33mm in the mid left circumflex artery (LCx).

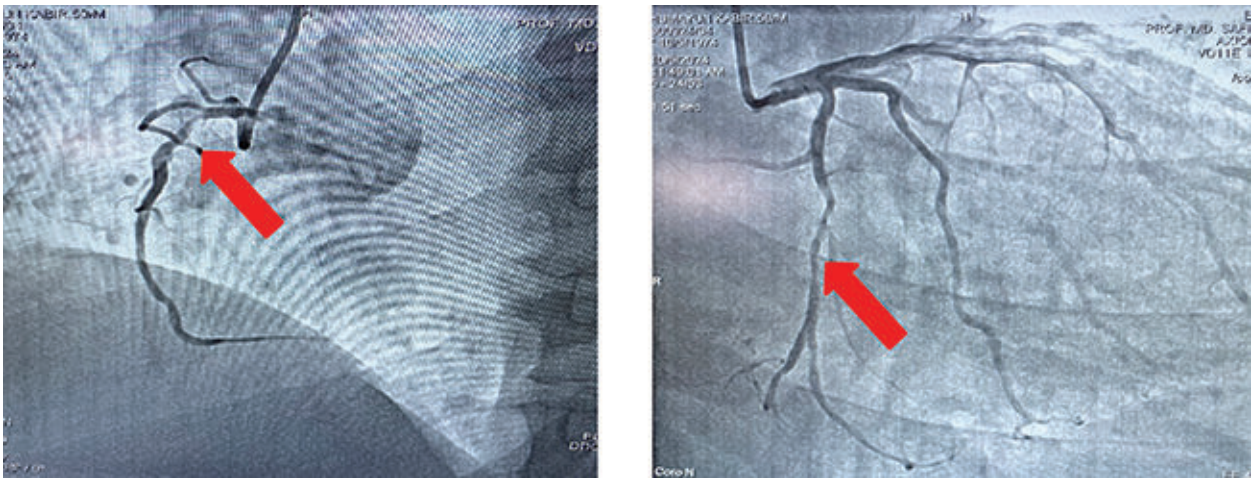

Figure 2: *CAG revealed 90-95% proximal stenosis in RCA & midsection of LCx exhibits 90% stenosis. Both has TIMI score 2flow.*

Troponin report chart:

30/09/2024	0.008ng/mL(popular diagnostic ltd)	AMI : 0.12 and above
30/09/2024	21.2 pg/mL (Cardiology Lab, BSMMU) (High sensitive)	00-60.4 pg/mL
1/10/2024	0.01ng/mL(laboratory medicine depot ,BSMMU)	0-0.1ng/mL
01/10/2024	27.200Pg/mL (High sensitive)(Cardiology Lab, BSMMU)	0.00-60.4Pg/mL
CK(MB) report Chart:		
30/09/2024	16U/L	7.00-25.00 U/L
01/10/2024	42.6U/L	<25U/L

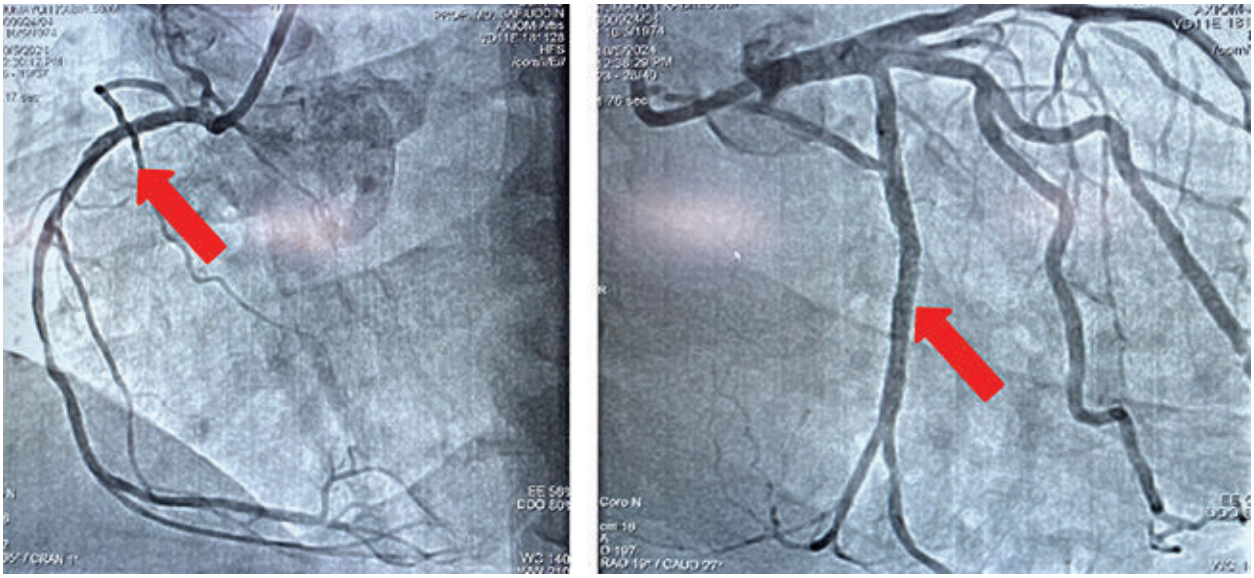

Figure 3: *After PTCA in RCA & LCx with TIMI 3 flow*

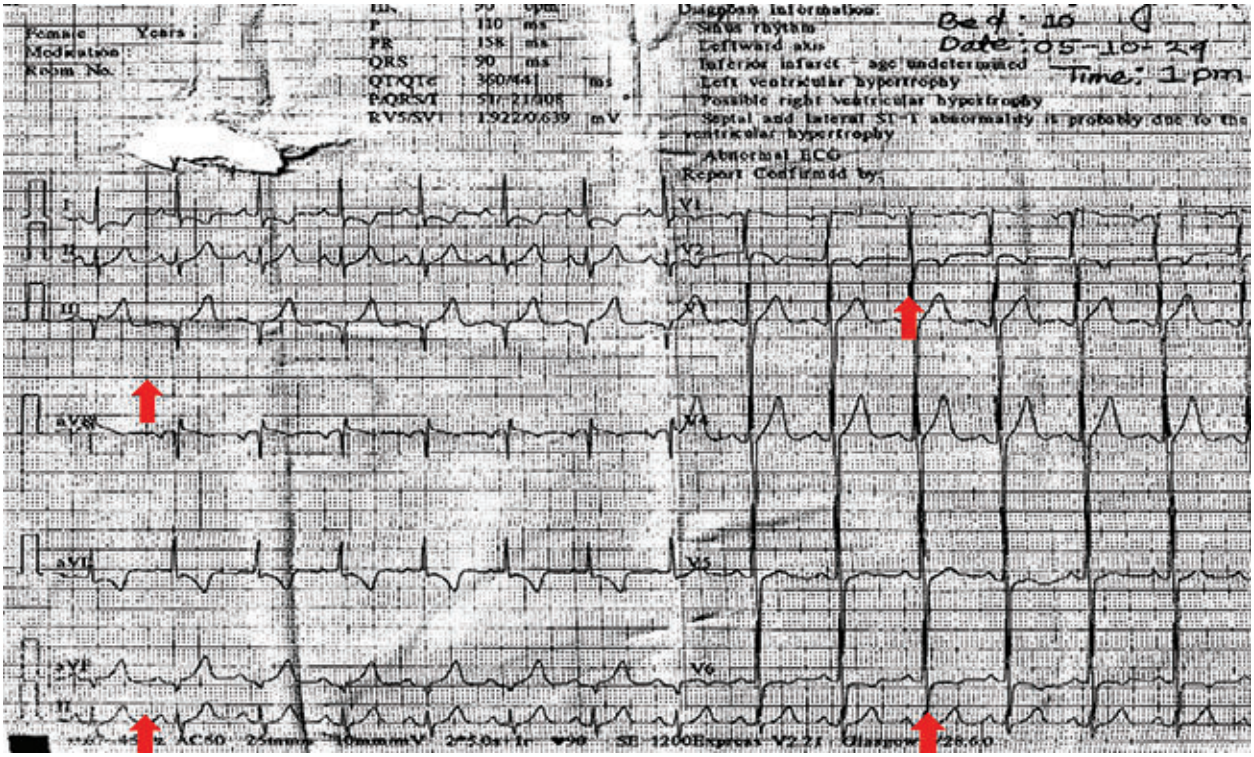

Figure 4: *ECG shows development of pathological Q wave lead III and aVF and T wave inversion in different leads.*

Discussion:

The diagnosis of acute coronary syndrome is based on the clinical feature of chest pain or discomfort, ST segment and T wave deviations on a 12-lead ECG, and changes in the serum concentrations of cardiac muscle enzymes mostly troponin I. The majority of individuals with ST segment elevation consistently have elevated serum cardiac muscle enzyme levels, signifying myocardial pathology (Mokhtari, A. et al., 2016). In 1965, researchers first identified troponins, and in the late 1990s, they established a reliable immunoassay for measuring their blood levels (Bodor, G.S., et al., 1995).

A part of the contractile machinery in cardiac and skeletal myocytes is troponin. The cardiac troponin complex consists of three isoforms: troponin C, troponin I, and troponin T. In cardiac myocytes, roughly 4%–5% of troponin is located in the cytosol, contributing to the initial increase of troponin upon myocardial damage. The residual troponin resides in the sarcomere, and upon cellular injury, it facilitates a gradual, sustained release of troponin over several days.

Troponin I and T exhibit more specificity for cardiac myocytes compared to troponin C; hence, they were established as the recommended biomarkers for myocardial damage (Danese, E. and Montagnana, M., 2016). In fact, CTnI has not been found anywhere other than the heart (Mokhtari,A. et al., 2016).

Troponin measures showed nearly 99% sensitivity when measured 6 to 12 hours post-onset of chest discomfort and demonstrated markedly enhanced specificity for cardiac muscle injury (Katus, H.A. et al., 1991). The American College of Cardiology incorporated serial troponin testing into the Fourth Universal Definition of Myocardial Infarction, citing its clinical utility (Garg, P., et al., 2017).

Numerous cardiac and non-cardiac pathologies can lead to elevated cardiac troponin levels concentration in blood. Acute Heart failure, cardiomyopathy, pulmonary embolism, arrhythmia, myocarditis, valvular heart disease, and cardiac contusions due to trauma are some of the conditions that can cause troponin levels to rise.

Noncardiac etiologies encompass renal failure, infections, anemia, hypotension, hypoxia, and noncardiac surgical procedures (Thygesen, K. et al., 2019). There are some other cause where false positive and false negative troponin result can be identified which are discussed below. (McCarthy, C.P., et al., 2019)

Our focus of attention are the false negative cases. Troponin concentrations may be influenced by hemolysis, resulting in both misleading elevations and reductions, depending on the assay (McCarthy, C.P., et al., 2019). Substantial hemolysis may lead to an inaccurately reduced cTn level (Garg, P., et al., 2017).

Conversely, hyperbilirubinemia (>10 gm/dl), cardiac troponin autoantibodies, and lipemia may diminish troponin concentration readings (Herman, D.S. et al., 2017).

Biotin (vitamin B7), a water-soluble vitamin within the B complex, found in OCT multivitamins for hair and skin, may also cause unnaturally low troponin levels (Masimasi, N. and Means Jr, R.T., 2005).

On the other hand, increased alkaline phosphatase level, Heterophile antibodies, fibrin level, rheumatoid factor (RA factor) interference has been linked to misleading elevations in troponin levels without definitive myocardial damage myopathies.

Regarding our case, analyzer malfunction was not possible because repeated troponin measurements before admission and during the hospital stay were negative. Troponin was initially done in popular diagnostic center then in Cardiology lab and then laboratory medicine department in BSMMU. All sample were negative. His liver function test was normal and there were no signs of hemolysis.

His lipid profile was also normal and no history of using over-the-counter medicine or vitamins. We actually could not find the cause of false negative result in our patient.

Conclusions:

Negative troponin level in a patient with STEMI is an uncommon occurrence, as this example demonstrates. One of the most sensitive markers of myocardial injury is cardiac troponin, which has a sensitivity value of more than 99 percent. On the other hand, it might be negative in the early stages of acute coronary syndrome and then return to normal. Some doubts remain regarding how our patient might have performed multiple tests and come back with a troponin-negative result. Physicians must be aware of this unusual finding.

Acknowledgments:

We want to thank our teammates of Dept of cardiology, BSMMU and all the clinical staffs who participated in the treatment of the patients in the hospital.

Funding information:

This study received no financing from any governmental, corporate, or non-profit organization.

Conflict of interest statement:

The authors state that they have no known conflicting financial interests or personal relationships that may be seen as having influenced the work described in this study.

Ethics statement:

As per our institutional ethics committee guidelines, we do not require ethics approval for publishing case reports.

Consent:

Written informed consent was obtained from the patient's

husband to publish this report including clinical facts and imaging in this article in accordance with the journal's patient consent policy

Reference:

1. Mokhtari, A., Lindahl, B., Smith, J.G., Holzmann, M.J., Khoshnood, A. and Ekelund, U., 2016. Diagnostic accuracy of high-sensitivity cardiac troponin T at presentation combined with history and ECG for ruling out major adverse cardiac events. Annals of Emergency Medicine, 68(6), pp.649-658
2. Herman, D.S., Kavsak, P.A. and Greene, D.N., 2017. Variability and error in cardiac troponin testing: an ACLPS critical review. American Journal of Clinical Pathology, 148(4), pp.281-295.
3. Masimasi, N. and Means Jr, R.T., 2005. Elevated troponin levels associated with hemolysis. The American journal of the medical sciences, 330(4), pp.201-203.
4. Kumar, A. and Cannon, C.P., 2009, October. Acute coronary syndromes: diagnosis and management, part I. In Mayo Clinic Proceedings (Vol. 84, No. 10, pp. 917-938). Elsevier.
5. McNeil, A., 2007. The trouble with troponin. Heart, Lung and Circulation, 16, pp.S13-S16.
6. Bodor, G.S., Porterfield, D., Voss, E.M., Smith, S. and Apple, F.S., 1995. Cardiac troponin-I is not expressed in fetal and healthy or diseased adult human skeletal muscle tissue. Clinical chemistry, 41(12), pp.1710-1715.
7. Danese, E. and Montagnana, M., 2016. An historical approach to the diagnostic biomarkers of acute coronary syndrome. Annals of translational medicine, 4(10)
8. Katus, H.A., Remppis, A., Scheffold, T., Diederich, K.W. and Kuebler, W., 1991. Intracellular compartmentation of cardiac troponin T and its release kinetics in patients with reperfused and nonreperfused myocardial infarction. The American journal of cardiology, 67(16), pp.1360-1367.
9. Garg, P., Morris, P., Fazlanie, A.L., Vijayan, S., Dancso, B., Dastidar, A.G., Plein, S., Mueller, C. and Haaf, P., 2017. Cardiac biomarkers of acute coronary syndrome: from history to high-sensitivity cardiac troponin. Internal and emergency medicine, 12, pp.147-155.
10. Thygesen, K., Alpert, J.S., Jaffe, A.S., Chaitman, B.R., Bax, J.J., Morrow, D.A. and White, H.D., 2019. Fourth universal definition of myocardial infarction (2018). European heart journal, 40(3), pp.237-269.
11. McCarthy, C.P., Raber, I., Chapman, A.R., Sandoval, Y., Apple, F.S., Mills, N.L. and Januzzi, J.L., 2019. Myocardial injury in the era of high-sensitivity cardiac troponin assays: a practical approach for clinicians. JAMA cardiology, 4(10), pp.1034-1042.