Junior researchers

ახალგაზრდა მკვლევარები

Vol. 4 Issue 2, 2026

https://doi.org/10.52340/jr.2026.04.02.18



High-Sensitivity Cardiac Troponins in the Emergency Department: Transforming Acute Coronary Syndrome Diagnosis

Parvin Mozafari[1], Anagha Krishna Devipriya[2], Shifa Tajuddin Syed[3], Mohammed Ammar Mahmoud Hijazi[4], Adam wazeeh[5], Sagi Murtada Nagm Eldin Abbas[6].

[1]Faculty of medicine, University of Georgia, Tbilisi, Georgia, Parvinmozaffari73@gmail.com, Phone number: +995-591096156. ORCID: 0009-0002-8107-2390; [2]Tbilisi State Medical University, Tbilisi, Georgia. ORCID: 0009-0007-2311-208X; [3]Tbilisi State Medical University, Tbilisi, Georgia. ORCID: 0009-0009-0400-5929; [4]Tbilisi State Medical University, Tbilisi, Georgia. ORCID: 0009-0001-3661-339X; [5]Tbilisi State Medical University, Tbilisi, Georgia. ORCID: 0009-0001-3651-5984; [6]Tbilisi State Medical University, Tbilisi, Georgia. ORCID: 0009-0002-9046-7079.

Abstract

Recent advances in emergency department (ED) assessment of suspected acute coronary syndrome (ACS) have been driven by the use of high-sensitivity cardiac troponin (hs-cTn) assays, which allow detection of myocardial injury at levels below the analytical detection limit of traditional cardiac troponin assays, and facilitate rapid diagnostic pathways. The value of these is, however, not to be judged based on the concentration of the biomarkers alone, but integrated with symptoms, electrocardiography (ECG), time of presentation, assay-specific thresholds, and serial change. This narrative review brings together literature provided by the authors regarding the evolution of hs-cTn assays, single-sample vs serial testing, point-of-care testing, sex-related differences, chronic kidney disease (CKD), hemodialysis, coronary CT angiography (CCTA), and risk stratification. We propose a pragmatic conceptual model in which hs-cTn helps with the rule-out, rule-in and observation decision-making process, but is not used as a standalone diagnosis of type 1 myocardial infarction (MI). Specific focus is placed on analytical and clinical constraints such as non-ACS elevations, inter-assay non-interchangeability, and uncertain generalizability of pathways to populations. Future directions include, but are not limited to, personalized interpretations, evidence-based incorporation of biomarker and anatomical imaging (with the support of artificial intelligence (AI) tools). Overall, it is concluded that when used as part of a structured clinical pathway, hs-cTn enhances the efficiency and sensitivity of diagnosis and should not be used in place of clinical reasoning or evidence of myocardial ischemia.

Keywords: high-sensitivity cardiac troponin, emergency department, acute coronary syndrome, myocardial infarction, point-of-care testing, serial measurement, chronic kidney disease.

Background

Acute coronary syndrome (ACS) continues to be a leading cause of mortality and morbidity in the world and encompasses clinical presentations related to acute myocardial ischemia, including myocardial infarction (MI). Prompt and accurate diagnosis of ACS in the ED is critical – time-poor diagnosis will mean time lost in treatment that could benefit from evidence-based therapies, and time-to-overy diagnosis will mean unnecessary admission, investigation, and utilization of resources. Clinical assessment and electrocardiography (ECG) are essential but may not rule in or out MI in all patients, including those with non-diagnostic ECG findings and atypical presentations. Reliable cardiac biomarkers are thus a crucial part of the contemporary diagnostic pathways. The biomarker concept of myocardial injury has undergone significant changes. Aspartate aminotransferase (AST) was initially used, followed by lactate dehydrogenase (LDH) and finally creatine kinase-MB (CK-MB). These markers were restricted in their tissue distribution, poor specificity of cardiac distribution and failed to consistently detect small injuries. Cardiac troponins (cTnT and cTnI) became the markers with higher specificity for myocardial injury and came to be the basis of the diagnosis of MI. However, traditional assays are less effective at detecting low levels of circulating factors early in the onset of symptoms, which may require more time for them to be observed and long-term sampling. High-sensitivity cardiac troponin assays (hs-cTn) were developed to increase analytical sensitivity and allow the detection of low levels of cardiac troponin, thus facilitating the implementation of shorter and faster algorithms for diagnosis [6,14,17].

Beyond a positive or negative value, the clinical use of hs-cTn goes beyond. Concentration at presentation and change over time can provide information for diagnostic and prognostic assessment. Low levels could be used to consider rule-out in select cases, and high levels are linked to myocardial injury and poor cardiovascular risk. In patients with comorbidities, serial measurements can be useful to differentiate an acute dynamic pattern from a persistently elevated baseline [2-4,16,19]. However, hs-cTn is not specific for ACS or plaque rupture. Other possible complications of myocardial injury with detectable or elevated troponin include myocarditis, heart failure, pulmonary embolism, sepsis, arrhythmia, hypoxia, and renal dysfunction [11,19]. Biological and clinical differences modify interpretation. In general, women tend to have lower reference concentrations than men and a single threshold may lead to under-detection of MI in women; the clinical implications and application of sex-specific thresholds will be assay- and context-specific [7,18]. Interpretation is complicated by the presence of CKD and hemodialysis since chronic myocardial injury or dialysis-associated changes may lead to either a persistent or fluctuating hs-cTn level [5,10,12].

Furthermore, both the hs-cTnT and hs-cTnI assays have varying analytical properties and decision limits and cannot be used interchangeably [9]. The diagnostic framework thus necessitates the separation of myocardial injury from MI. If the hs-cTn level is elevated, then there is myocardial injury, but if the level is normal, then there is no clinical evidence of myocardial injury. A rise and/or fall pattern helps to identify acuity but should be assessed with ischemic symptoms, ECG and other clinical evidence. This is especially true for the ED, where the early presenter may not have had an increase in

concentration, and the patient with a chronic disease may have a higher baseline concentration [2,4,5,11]. Rapid pathways are now routinely available that include repeat testing at set intervals, clinical risk assessment and ECG as well as hs-cTn at the start. Point-of-care (POC) hs-cTn can shorten turnaround times and aid triage, and selected patients with intermediate risk, after initial assessment, can benefit from the additional information provided by CCTA regarding coronary anatomy [1,8,15]. Neither technology should be applied as standalone: thresholds and quality control must be per assay; patient selection is necessary and integration with validated local protocols is required.

In this context, this narrative review aims to explore the evolution of the use of hs-cTn in the ED setting for suspected ACS, highlighting the use of accelerated testing, serial measurement, clinical interpretation, patient-specific modifiers and the incorporation of POC testing, CCTA, risk scores and novel AI strategies. It also addresses current debates and research agendas that could influence more specific and fair diagnostics-based care.

Methodology

This article is a narrative review, and it is based on the reference list provided by the authors as a primary evidence base. Included in the provided bibliography were 20 publications, all of which involved original cohort studies, diagnostic accuracy studies, systematic reviews, meta-analyses, or narrative reviews of the use of hs-cTn in the ED evaluation of patients with suspected ACS. The source set was reviewed against the review objectives and organized into thematic domains: (1) Evolution and analytical/clinical performance of hs-cTn; (2) single-sample and serial rule-out/rule-in strategies; (3) POC hs-cTn; (4) Interpretation in sex, age, CKD, and hemodialysis contexts; (5) Myocardial injury versus MI and non-ACS elevations; (6) CCTA and risk stratification; (7) AI, implementation and knowledge gaps. The qualitative synthesis of evidence was conducted instead of quantitative. Results were compared by study design and clinical focus, and included diagnostic sensitivity, negative predictive value, pathway efficiency, interpretive limitations, and applicability to ED practice.

The references that have been given below have been numbered as per the bibliography of the original paper. This is not a systematic review because no prospective protocol, comprehensive database search, risk of bias assessment, or meta-analysis has been done. Therefore, the discussion is an interpretation of the review and should be taken only as a narrative review of the literature that has been provided and not a systematic review of the evidence. Numerical statements or assays provided by source papers are only for the purpose of observation and cannot be taken as universal limits. Decision limits and sampling intervals must be verified for the assay and clinical pathway. The bibliographic details in the list provided below should be verified from publisher or indexed records.

Discussion

Hs-cTn as a sensitive marker within the ACS diagnostic pathway

Hs-cTn improves the diagnosis of minor myocardial damage and allows an earlier classification of patients with suspected ACS. Clinical performance is usually quantified in terms of sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV). NPV and sensitivity are especially valuable for ruling out disease, although PPV and NPV are conditional upon prevalence of the disease and the choice of the population. Therefore, a low level should not be seen as one of the criteria in itself to exclude MI, but rather as [3,4,17].

This is one conceptual change: The simple single cutoff is replaced by the complex pathway. Stage 1 includes history, onset of symptoms, an ECG and a test for hs-cTn. Very minimally involved patients may have a chance to be ruled out; the high-risk patients or those who show ischemia signs or features should receive further investigations. See Figure 1. This conceptual approach is not a replacement for locally validated assay specific algorithms.

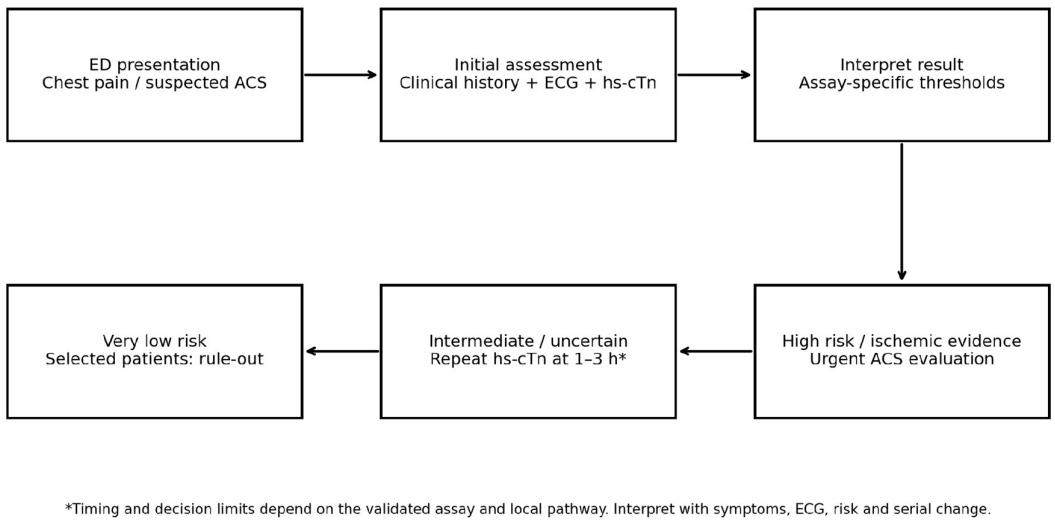


Figure 1. Conceptual ED diagnostic pathway integrating clinical assessment, ECG, hs-cTn, and risk-directed follow-up. The diagram is illustrative; thresholds and intervals must follow validated assay-specific protocols.

Single-sample testing versus serial measurement

In some patients, one positive very-low hs-cTn test can contribute to early rule-out, particularly if the onset of symptoms is distant and the clinical picture is reassuring. The studies on single-sample strategies have focused on their ability to decrease observation and increase disposition [1,4]. A single sample, however, may not be enough for early presenters or patients with persistent symptoms or clinical risk. Central to the interpretation is the release kinetics of troponin and the time delay between

the onset of symptoms and the blood sample collected. In serial sampling (usually carried out in a 0/1-hour, 0/2-hour or 0/3-hour pathway depending on the assay and protocol) absolute concentration and change are evaluated. A rapid increase and/or decrease is associated with acute myocardial damage while a more gradual increase is associated with chronic myocardial damage. The changes and decision limits vary by assay and cannot be extrapolated for any other platform [2,17]. The concept of dynamic versus relatively stable trajectories is shown as a conceptual illustration in Figure 2.

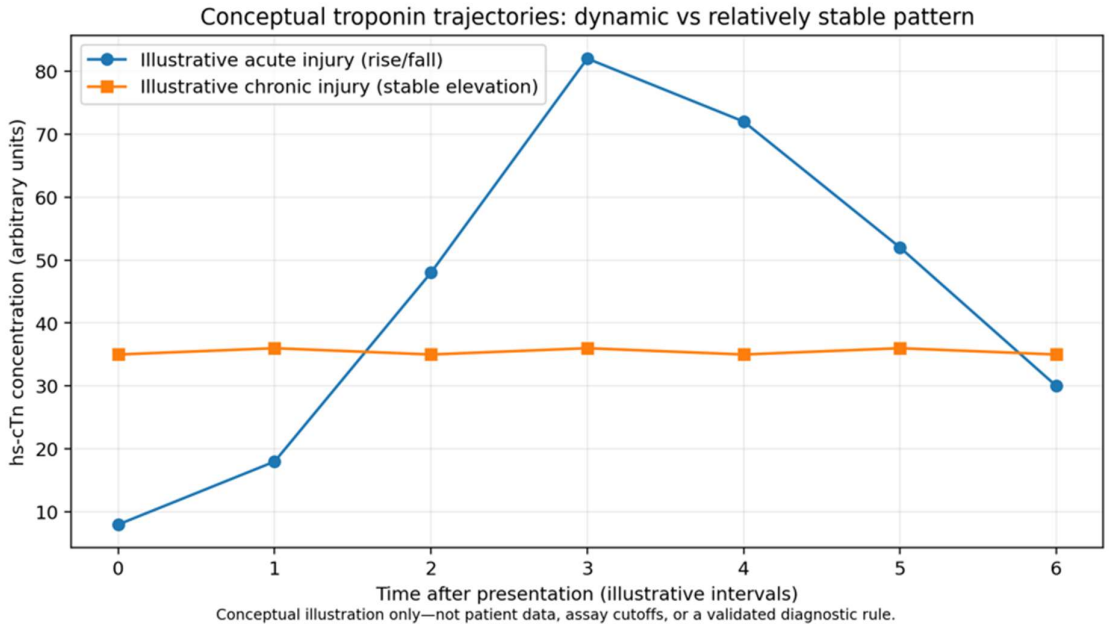


Figure 2. Conceptual comparison of dynamic acute injury and relatively stable chronic myocardial injury trajectories. Curves are illustrative only and do not represent real patient data or diagnostic cutoffs.

Myocardial injury is not synonymous with MI

One of the major limitations of hs-cTn is that it detects myocardial injury but not the mechanism of injury. The acute myocardial injury plus clinical evidence of acute ischemia is a requirement for the diagnosis of MI. Troponin can be raised in heart failure, myocarditis, cardiomyopathy, hypertensive heart disease, pulmonary embolism, sepsis, stroke, anemia, hypoxia and shock, and arrhythmias. In these contexts, injury might occur due to imbalance of supply and demand, hemodynamic stress, or other mechanisms without the presence of acute coronary atherothrombosis [11,19]. This higher analytical sensitivity translated into greater numbers of patients with injury being identified, and a greater reliance on clinical interpretation. Positive biomarker should lead towards assessment of the cause and not immediately to diagnosis of ACS. It is important to consider symptoms, ECG, the hemodynamic status, and the course of hscTn, if available, alongside.

Sex-associated differences and threshold selection

Circulating levels of hs-cTn are different in the two sexes, which may have implications for diagnostic thresholds. Lower reference concentrations may be used for women and use of high-sensitivity assays and sex-specific thresholds may detect further women with MI who may not otherwise have been detected [7,18]. However, threshold selection can also be complicated – the value of implementation is also dependent on the assay, the reference population, and the clinical response at the downstream level. However, just increasing detection does not lead to better outcomes, unless diagnosis is followed by appropriate investigation, treatment and secondary prevention.

CKD, hemodialysis, and age-related interpretation

The main difficulty with this population is that the level of hs-cTn can be chronically elevated, suggesting chronic myocardial injury and coexisting cardiac structural disease. It can be caused by decreased renal function, inflammation, anemia, hypertension, ventricular hypertrophy and vascular disease. The one elevated level is therefore not as specific for acute MI and serial levels, symptoms, ECG and overall clinical picture become essential [5,12,19].

Timing and modality of hemodialysis may also impact measured hs-cTn. Dialysis should not take place at an arbitrary time of the day, and the concentration should not be taken as a stable baseline [10]. Older age is also often associated with cardiovascular disease and chronic injury; and should be taken into consideration in the clinical context but should not be used to negate raised results and should not be used to set and accept higher reference levels that are not validated. In these populations, an absolute concentration and a dynamic change is more convenient than individual threshold reasoning.

POC hs-cTn: speed, performance, and implementation

POC hs-cTn could reduce the time from sample collection to results, which may positively impact ED flow and prompt decision making. Selected POC platforms appear to have good diagnostic performance but analytical sensitivity, precision around low concentrations, cutoffs, type and quality of specimens tested and quality-control requirements vary [1,15]. All of this should be integrated into a clinical pathway that is governed by the operator's training, calibration, quality assurance, and clear escalation policy and should be called POC testing. A quick result is of little value if it is not analytically valid and clinically interpreted.

CCTA and clinical risk stratification

The biochemical evidence of myocardial injury is provided by Hs-cTn, while the coronary anatomy (presence of plaques and stenosis) is assessed by CCTA. In some patients with a moderate risk initial ECG and hs-cTn, the modalities can be used alongside each other. There is no absolute indication for CCTA in all patients who have been safely excluded by a validated pathway, it should be considered in the context of radiation exposure, contrast use, renal status, coronary calcium, heart rate, availability, and local experience [8]. The HEART score and EDACS are risk scores that may be used to give extra clinical context since they incorporate variables that are not detected by troponin. Risk stratification

should be used to complement, not replace, clinical judgment. Patients with low risk can be discharged as per an appropriate pathway, patients with intermediate risk should have repeat hs-cTn and imaging should be considered, patients with high risk should receive prompt evaluation and management. Implementation studies highlight the importance of pathways being adopted into the context of the real-world care system for their performance and their effectiveness [16].

AI, individualized interpretation, and future directions

AI and machine-learning algorithms could be used to integrate hscTn concentrations and deltas with ECG findings, time of symptoms, age, gender, renal function, previous coronary disease and clinical risk factors to calculate individual risks for MI. These could go beyond dichotomous threshold biomarkers. But models need external validation, transparent reporting, calibration and subgroup bias assessment and prospective clinical outcome assessment. Reduced missed MI, avoidable admissions, ED LOS and cost are unknown if an AI-supported pathway is implemented based on diagnostic discrimination alone. Personalized thresholds, which include sex, age, renal function and baseline cardiovascular status, are also being actively investigated. A future strategy that is likely to be practical will involve using validated algorithms, dependent on the specific assay, and patient context, in addition to selective anatomical imaging. Harmonization between platforms, robust evidence in CKD and dialysis, safety of early presenters, equity across demographic groups, POC implementation, prospective evaluation of patient-centered outcomes are priority areas.

Knowledge gaps and controversies

There are several questions that remain unanswered: What is the best balance between ease of testing with a single sample and safety of serial testing? Do sex-specific thresholds consistently yield better clinical results? How to optimize interpretation in CKD and dialysis patients? How to compare different assays of hs-cTnI with different assays of hs-cTnT? What intermediate-risk patients are best suited for CCTA? And does adding AI to existing pathways result in better outcomes? Another consideration is generalizability, the performance of the pathway can change as disease prevalence changes, population characteristics change, assays are made available, or healthcare systems change and it is important to keep these factors in mind [5,7,8,9,15-19]. The evidence presented for this review has varied design, population, and the narrative approach does not allow for formal quantitative comparison. Moreover, numerical thresholds and sampling points are to be considered pathway specific. All this all but confirms the necessity of taking precautions when synthesizing and validating locally.

Conclusion

The high-sensitivity cardiac troponin test revolutionized our approach to diagnosing possible ACS in the emergency department through the detection of myocardial injury and the ability for rapid rule-out and rule-in protocols. The most useful information can be derived from interpretation of the test in the context of the symptom presentation, the ECG findings and the patient's clinical risk. hs-cTn is a marker for myocardial injury and not specific for MI on its own. There is a need to understand the difference between acute injury, chronic injury, and ischemic infarction. A one-sample method can be

used to hasten disposition among suitable candidates, while serial sampling can be used among acute presenters, indeterminate results, persistently concerning patients, and CKD patients, among others.

POCT could enhance turn-around times, and CCTA could provide additional complementary anatomic details for certain intermediate-risk patients. Differences related to sex, kidney disease, hemodialysis, age, and specific assay-related issues demand personalized but evidence-based evaluation.

The future of further advancements relies on assay-specific validated algorithms, correct use of technology, more harmonization, and prospective evaluation of novel personalized medicine and AI-based approaches. Ultimately, hs-cTn needs to be regarded as a potent element of comprehensive clinical decision-making process, not a standalone diagnostic end point.

Table 1. Overview of hs-cTn strategies in ED evaluation of suspected ACS

Strategy	Main clinical role	Strengths	Important limitations / cautions
Single-sample hs-cTn	Rule-out in carefully selected patients	Potentially rapid disposition; fewer repeat samples [1,4]	Early symptom onset, ongoing symptoms, or non-low clinical risk may require serial testing
Serial hs-cTn (e.g., 0/1, 0/2, 0/3 h)	Assess concentration and dynamic change	Supports assessment of acute vs chronic injury [2,17]	Timing and delta criteria are assay- and pathway-specific
POC hs-cTn	Rapid bedside result and triage	Can reduce turnaround time [1,15]	Platform variation; quality control, training, and validation required
hs-cTn plus clinical risk score	Risk-directed disposition	Adds history and risk variables beyond biomarker [16]	Score performance may vary by population and care setting
hs-cTn plus selective CCTA	Intermediate-risk anatomical assessment	Provides coronary plaque/stenosis information [8]	Radiation, contrast, renal function, calcification, availability

Table 2. Clinical factors that influence hs-cTn interpretation

Factor / context	Potential influence	Interpretive approach
Early presentation after symptom onset	Troponin may not yet have risen	Consider serial testing and symptom timing [2,4,17]
CKD	Baseline hs-cTn may be chronically elevated	Use clinical context, ECG, serial change; avoid diagnosing MI from one value [5,12]
Hemodialysis	Concentration may vary with dialysis timing	Document timing and interpret serial values cautiously [10]
Sex-associated differences	Reference concentrations may differ	Consider validated assay-specific sex thresholds and clinical context [7,18]
Older age / comorbidity	Chronic injury and baseline elevation more frequent	Avoid dismissing elevations; assess trajectory and ischemic evidence
Non-ACS illness (e.g., sepsis, PE, HF, myocarditis)	Myocardial injury may occur without type 1 MI	Evaluate cause of injury and evidence of ischemia [11,19]
hs-cTnI versus hs-cTnT platform	Analytical characteristics and thresholds differ	Do not interchange numeric values or cutoffs [9]

Table 3. Complementary tools and future directions

Tool / development	Contribution	Key considerations
ECG and clinical assessment	Evidence of ischemia; clinical probability	Essential context for biomarker interpretation
HEART / EDACS risk assessment	Structured risk estimate	Use within validated local protocols
CCTA	Coronary anatomical information	Select for patients likely to benefit; consider contrast and radiation [8]
AI / machine learning	Multivariable individualized risk estimation	External validation, transparency, calibration, subgroup bias, outcome evidence
Personalized thresholds	Potentially account for sex, age, renal function, baseline risk	Evidence and standardization remain incomplete
POC pathway optimization	Faster turnaround and triage	Quality assurance and platform-specific validation [15]

Acknowledge

The authors confirm that all individuals listed as authors made substantial, equal contributions to the development of this work, including the abstract, introduction, methodology, discussion, and conclusion. The collaborative nature of this project reflects the shared responsibility and joint effort of all team members.

References

1. Apple FS, Smith SW, Greenslade JH, Sandoval Y, Parsonage W, Ranasinghe I, et al; SAMIE Investigators. Single high-sensitivity point-of-care whole-blood cardiac troponin I measurement to rule out acute myocardial infarction at low risk. Circulation. 2022;146(25):1918-29. doi:10.1161/CIRCULATIONAHA.122.061148.

2. Kavsak PA, Hewitt MK, Mondoux SE, Cerasuolo JO, Ma J, Clayton N, et al. Diagnostic performance of serial high-sensitivity cardiac troponin measurements in the emergency setting. J Cardiovasc Dev Dis. 2021;8(8):97. doi:10.3390/jcdd8080097.

3. Chapman AR, Lee KK, McAllister DA, Cullen L, Greenslade JH, Parsonage W, et al. Association of high-sensitivity cardiac troponin I concentration with cardiac outcomes in patients with suspected acute coronary syndrome. JAMA. 2017;318(19):1913-24. doi:10.1001/jama.2017.17488.

4. Fabre-Estremera B, Smith SW, Sandoval Y, Schulz K, Okeson B, Cullen L, Apple FS. Rapid rule-out of myocardial infarction using a single high-sensitivity cardiac troponin I measurement strategy at presentation to the emergency department: the SAFETY study. Clin Chem. 2023;69(6):627-36. doi:10.1093/clinchem/hvad033.

5. Chuang AM, Nguyen MT, Kung WM, Lehman S, Chew DP. High-sensitivity troponin in chronic kidney disease: considerations in myocardial infarction and beyond. Rev Cardiovasc Med. 2020;21(2):191-203. doi:10.31083/j.rcm.2020.02.17.

6. Dalal JJ, Ponde CK, Pinto B, Srinivas CN, Thomas J, Modi SK, et al. Time to shift from contemporary to high-sensitivity cardiac troponin in diagnosis of acute coronary syndromes. Indian Heart J. 2016;68(6):851-5. doi:10.1016/j.ihj.2016.03.013.

7. Ramos HR, López LE, Quiroga Castro W, Serra CMJ. High-sensitivity cardiac troponins: sex-specific values in clinical practice. Precision or confusion? Hellenic J Cardiol. 2019;60(3):171-7. doi:10.1016/j.hjc.2019.02.005.

8. Yang S, Manjunath L, Willemink MJ, Nieman K. The role of coronary CT angiography for acute chest pain in the era of high-sensitivity troponins. J Cardiovasc Comput Tomogr. 2019;13(5):267-73. doi:10.1016/j.jcct.2019.05.007.

9. Perrone MA, Storti S, Salvadori S, Pecori A, Bernardini S, Romeo F, et al. Cardiac troponins: are there any differences between T and I? J Cardiovasc Med (Hagerstown). 2021;22(11):797-805. doi:10.2459/JCM.0000000000001155.

10. Hunderup MM, Kampmann JD, Kristensen FB, Hafsund M. The short-term effect of hemodialysis on the level of high-sensitive cardiac troponin T: a systematic review. Semin Dial. 2024;37(2):110-1. doi:10.1111/sdi.13178.

11. Chaulin AM. False-positive causes in serum cardiac troponin levels. J Clin Med Res. 2022;14(2):80-7. doi:10.14740/jocmr4664.

12. Geladari EV, Vallianou NG, Evangelopoulos A, Koufopoulos P, Panagopoulos F, Margellou E, et al. Cardiac troponin levels in patients with chronic kidney disease: markers of high risk or just noise? Diagnostics (Basel). 2024;14(20):2316. doi:10.3390/diagnostics14202316.

13. Lee MMY, Masri A. Differentiating cardiac troponin levels during cardiac myosin inhibition or cardiac myosin activation treatments: drug effect or the canary in the coal mine? Curr Heart Fail Rep. 2023;20(6):504-18. doi:10.1007/s11897-023-00620-2.

14. Garg P, Morris P, Fazlanie AL, Vijayan S, Dancso B, Dastidar AG, et al. Cardiac biomarkers of acute coronary syndrome: from history to high-sensitivity cardiac troponin. Intern Emerg Med. 2017;12(2):147-55. doi:10.1007/s11739-017-1612-1.

15. Blanc L, Vaissaire A, Renard N, Vargmo C, Størvold GL, Bouhadef A, Claret PG. Analytical and clinical performance of high-sensitivity cardiac troponin point-of-care assays as an aid in the diagnosis of myocardial infarction: a narrative review. Emerg Med Int. 2025;2025:5717892. doi:10.1155/emmi/5717892.

16. McDermott M, Kimenai DM, Anand A, Huang Z, Houston A, Williams S, et al; HDRUK Regional Linked Data Driven Evidence Network. Adoption of high-sensitivity cardiac troponin for risk stratification of patients with suspected myocardial infarction: a multicentre cohort study. Lancet Reg Health Eur. 2024;43:100960. doi:10.1016/j.lanepe.2024.100960.

17. Westwood ME, Armstrong N, Worthy G, Fayter D, Ramaekers BLT, Grimm S, et al. Optimizing the use of high-sensitivity troponin assays for the early rule-out of myocardial infarction in patients presenting with chest pain: a systematic review. Clin Chem. 2021;67(1):237-44. doi:10.1093/clinchem/hvaa280.

18. Shah AS, Griffiths M, Lee KK, McAllister DA, Hunter AL, Ferry AV, et al. High sensitivity cardiac troponin and the under-diagnosis of myocardial infarction in women: prospective cohort study. BMJ. 2015;350:g7873. doi:10.1136/bmj.g7873.

19. Maayah M, Grubman S, Allen S, Ye Z, Park DY, Vemmou E, et al. Clinical interpretation of serum troponin in the era of high-sensitivity testing. Diagnostics (Basel). 2024;14(5):503. doi:10.3390/diagnostics14050503.

20. Capodifoglio S, De Iuliis V. Diagnostic accuracy of point-of-care high-sensitivity troponin algorithms for emergency department rule-out and rule-in of acute myocardial infarction: a systematic review and meta-analysis. Emerg Med J. 2026. doi:10.1136/emermed-2026-215922.