

Rapid troponin algorithms in Emergency Care: from biomarker to clinical practice

Algoritmos rápidos de troponina en urgencias: del biomarcador a la práctica clínica

Aitor Alquézar-Arbé¹, Beatriz López Barbeito²

Non-traumatic chest pain is the second most frequent reason for consultation in emergency departments (EDs).¹ Although the differential diagnosis of this entity includes potentially life-threatening conditions requiring immediate attention, in most cases the cause is a non-cardiac process. In fact, only between 5–10 % of patients evaluated for non-traumatic chest pain in the emergency setting are ultimately diagnosed with myocardial ischemia, whereas more than half are classified as having nonspecific pain or pain of non-cardiac origin.²

The introduction of high-sensitivity cardiac troponin (hs-cTn) has significantly transformed the diagnosis of non-ST-segment elevation acute coronary syndrome (NSTEMI). However, it has also generated new interpretative challenges. Currently, it is no longer sufficient to simply “order a troponin”: the real challenge lies in its correct interpretation within an uncertain clinical context, in a high-pressure care environment such as the ED, and with the frequent imprecision of information, both regarding symptom onset timing and the timing of hs-cTn measurement. European guidelines have been clear in this regard: the value of hs-cTn does not lie in an isolated measurement, but in its integration into rapid and protocolized 0/1 h or 0/2 h algorithms.³ Historically, troponin was considered a biochemical marker to confirm or rule out acute myocardial infarction (AMI). However, when using rapid hs-cTn algorithms, it is crucial to bear in mind that they detect the presence of myocardial injury, but do not indicate its etiology; that is, they do not diagnose AMI by themselves.⁴

According to the 4th universal definition of myocardial infarction, myocardial injury is defined by the presence of hs-cTn values > 99th percentile of the upper reference limit. This injury is considered acute when there is a dynamic pattern of rise and/or fall in troponin values, whereas it is considered chronic when values remain persistently elevated without

significant changes in serial measurements. In addition to AMI, the diagnosis of AMI requires demonstration of clinical evidence of myocardial ischemia, whether through compatible symptoms, electrocardiographic changes suggestive of ischemia, evidence of loss of viable myocardium on imaging studies, or identification of a coronary thrombus. Furthermore, a distinction must be made between type 1 and type 2 AMI. Type 1 AMI results from an acute atherothrombotic event due to rupture or erosion of an atherosclerotic plaque with coronary thrombus formation, whereas type 2 AMI is due to an imbalance between myocardial oxygen supply and demand not related to acute atherothrombosis, as occurs in situations such as severe anemia, sustained tachyarrhythmias, hypotension, or hypoxemia.

Consequently, the algorithms allow rapid identification of the presence of acute myocardial injury. However, distinguishing between type 1 AMI, type 2 AMI, or other causes of myocardial injury necessarily requires integration of the clinical context, electrocardiogram findings, and, in certain cases, imaging results.⁵ Finally, the diagnosis of unstable angina should be reserved for patients with evidence of myocardial ischemia (compatible symptoms and/or suggestive electrocardiographic findings), but without AMI; that is, without elevation of hs-cTn.⁶

In this context, the work by De la Fuente García *et al.*⁷ provides a particularly illustrative view of real-world practice in our setting. This is a retrospective study that does not evaluate the diagnostic performance of the algorithms, but rather something perhaps more relevant: their actual degree of implementation in the ED of a Spanish university hospital. The most striking finding of the study does not lie in extreme hs-cTn values, which behave as expected, but in the intermediate group. Of 157 patients classified in the observation zone (3–120 ng/L), only 12 underwent a second determination within the recom-

Author Affiliations:

¹Servei de Urgències, Hospital de la Santa Creu i Sant Pau, Barcelona, Spain.

²Àrea d'Urgències, Hospital Clínic, Barcelona, Spain.

Corresponding Author:

Aitor Alquézar-Arbé, Servei de Urgències, Hospital de la Santa Creu i Sant Pau, Carrer de Sant Quintí, 89, 08041 Barcelona, Spain.

E-mail:

aitor76px@hotmail.com

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mended interval. In other words, the true core of the algorithm—protocolized reassessment—was practically not applied. The clinical consequence of this omission is not merely theoretical. Two patients from this group returned days later with hs-cTn levels > 10,000 ng/L, requiring admission to intensive care. This is not a statistical finding, but a tangible example of how failure to apply the algorithm can have a direct clinical impact.

In addition to the small number of patients in whom serial hs-cTn measurements were performed, it should be noted that diagnostic coding was based on the final diagnosis recorded in the electronic health record. Although there is no international reference document standardizing diagnostic adjudication methodology in this type of study, common practice in many previous works involves final diagnosis adjudication by 2 independent investigators—not by the physician who provided patient care—using all available clinical information. In cases of discrepancy, a 3rd investigator is consulted.^{8,9} Although the approach adopted by the authors is more consistent with real-world clinical practice, it may have led to inappropriate diagnostic assignments. Furthermore, as a consequence of the above, the diagnostic categories used in this study differ from those conventionally accepted. Specifically, the classification of AMI (encompassing NSTEMI and ST-segment elevation myocardial infarction) and non-AMI cardiac disease (including unstable angina, arrhythmias, heart failure, hypertensive urgency, among others) is unusual in this type of research. First, ST-segment elevation myocardial infarction should not be included, as its management is emergent and its diagnosis is not based on hs-cTn determination.¹⁰ Moreover, some of the listed conditions, such as

arrhythmias, should be classified as type 2 AMI if the patient presents acute myocardial injury in a clinical context of ischemia.⁴ These 2 aspects should be taken into consideration when interpreting the results of the present study.

What this study demonstrates, without explicitly intending to do so, is that the problem does not lie in the analytical capacity of hs-cTn, nor in the validity of the decision thresholds proposed in the algorithms. The real challenge lies in their correct implementation and appropriate application in real clinical practice. In this regard, it is recommended that hospitals establish consensus protocols among EDs, cardiology, and the clinical laboratory.¹¹

Finally, it is likely that in the coming years clinical practice will incorporate artificial intelligence (AI) tools to assist in medical decision-making. Currently, there are already AI-based algorithms designed for the diagnosis of AMI based on ECG interpretation¹² and others that combine clinical, electrocardiographic, and hs-cTn parameters.^{13,14}

Despite the methodological limitations mentioned, the main value of this work lies in highlighting the gap between available scientific evidence and its implementation in real clinical settings. Multiple studies have demonstrated the ability of rapid hs-cTn-based algorithms to expedite diagnosis and improve risk stratification in patients with suspected acute coronary syndrome. However, their effectiveness largely depends on their effective integration into ED workflows. Reducing this gap between evidence and practice likely constitutes one of the most important challenges for emergency medicine in the coming years, and studies such as the one published in *Rev Esp Urg Emerg* help identify the critical points on which these efforts should be focused.

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