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Unselected cardiac troponin testing and the diagnosis of myocardial infarction in the emergency department

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Abstract

Background This research examines the role of systematic cardiac troponin evaluation in identifying type 1 myocardial infarction among patients presenting to the emergency department with collected blood samples.

Methods This was a prospective study of consecutive adult patients presenting to the emergency department of a university hospital between October 22, 2020, and January 11, 2021. Cardiac troponin I levels were measured in all patients, including those with suspected acute coronary syndrome (clinical testing) and a control group undergoing routine blood tests (non-clinical testing). The primary outcomes were the prevalence of type 1 myocardial infarction and the positive predictive value of cardiac troponin I, which were assessed using established statistical methods.

Results Elevated cardiac troponin levels were identified in 13.4% of the study population (382/2,853). This included 19.5% of patients with clinically guided tests and 10.1% of those with non-clinical testing. The overall prevalence of type 1 myocardial infarction was 2%, with a positive predictive value of 14.9% (95% CI: 13.6–16.2). Among clinically guided tests, type 1 myocardial infarction prevalence was 5.8%, yielding a positive predictive value of 29.5% (95% CI: 26.7–32.4). Cases from non-clinically guided tests were primarily attributed to type 2 myocardial infarction or non-ischemic myocardial injury.

Conclusion Using a generalized approach to cardiac troponin testing in emergency department patients significantly lowers the diagnostic accuracy for type 1 myocardial infarction, reducing the positive predictive value and frequently indicating non-ischemic myocardial injury.

Keywords Cardiac troponin, Myocardial infarction, Emergency department

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Introduction

Cardiac troponin (cTn) testing is fundamental in diagnosing myocardial infarction among patients in emergency departments [1]. While advances in cTn assays have increased their sensitivity, this improvement has often come at the expense of specificity [2–4]. The evolution of high-sensitivity cTn assays has greatly enhanced the detection of myocardial infarction, including both type 1 (T1MI) and type 2 (T2MI), as well as non-ischemic myocardial injury (NIMI) [5, 6]. However, this increased detection has also led to challenges in distinguishing between ischemic and non-ischemic conditions [7–9].

Although chest pain is a common symptom in patients with myocardial infarction, other clinical manifestations (dyspnea, syncope, etc.) may also be present [10]. Given that clinicians must maintain a high degree of suspicion in patients with atypical presentations, the request for cTn varies significantly among different emergency departments [11–13]. In many of these departments, patients undergo simultaneous testing for both cardiac and non-cardiac conditions to facilitate early diagnosis or discharge [14]. This increased use of cTn, in an often low-pretest probability setting for infarction, reduces the positive predictive value when elevated troponin levels are detected [8]. Using high-sensitivity troponin tests without a targeted clinical context can lead to increased diagnostic ambiguity [15, 16].

This study aims to assess the occurrence of myocardial injury in emergency department patients and to evaluate how broad use of cardiac troponin testing impacts diagnostic accuracy for myocardial infarction.

Methods

Study population

We conducted a prospective study including consecutive patients aged 18 years and older who visited the emergency department at Joan XXIII University Hospital in Tarragona between October 22, 2020, and January 11, 2021. This hospital serves as a general basic hospital for an area with a reference population of approximately 200,000 inhabitants and as a tertiary cardiology referral center for 800,000 inhabitants.

All patients for whom the attending physician requested cTn measurement, typically as a result of suspected acute coronary syndrome, were included, along with all patients who had blood samples taken for any reason other than suspected acute coronary syndrome, where cTn had not been requested. This troponin determination was performed on the excess serum from blood samples retained by the central laboratory for safety reasons or for repeat testing if necessary. These results were not disclosed to the attending physician to guide clinical care. The study was approved by our Institutional Review Board (*Comité Ético de Investigación con Medicamentos del Institut d'Investigació Sanitària Pere Virgili*) and conducted in accordance with the Declaration of Helsinki. The approval registry number is CEIM 195/2020. Since the leftover material was acellular, the ethics committee determined that individual patient consent was not required. Patients residing outside the study's reference area were excluded.

Troponin assay

Cardiac troponin I (cTnI) levels were assessed using an immunoassay method (Siemens Advia Centaur, USA) with detection limits set at 2.5 ng/L for the lower range and 25,000 ng/L for the upper range, as specified by the manufacturer. The reference limit for cTnI positivity was > 47 ng/l (corresponding to the 99th percentile with a total analytical imprecision, expressed by the coefficient of variation, < 10%), which was considered as elevated cTnI or myocardial injury.

Clinical variables studied

The electronic medical records of all the patients were reviewed. Demographic variables, cardiovascular risk factors, relevant cardiovascular and non-cardiovascular history, physical examination findings during the initial emergency evaluation, electrocardiographic findings, and laboratory tests were included. The glomerular filtration rate was calculated using the Chronic Kidney Disease Epidemiology Collaboration formula. Whether the patient was hospitalized or discharged and the primary diagnoses were also recorded. Myocardial infarction types (T1MI and T2MI) and non-ischemic myocardial injury (NIMI) were categorized following the Fourth Universal Definition of Myocardial Infarction, based on a consensus reached by two cardiologists [17], following the criteria proposed by Saaby et al. [18].

Primary and secondary outcome events

The study's primary outcome was the prevalence of T1MI and the positive predictive value of cTnI in the total population as well as in the population where troponin was requested based on clinical criteria.

Statistical analysis

Data for categorical variables were summarized using frequencies and percentages, while continuous variables were represented as medians with interquartile ranges. Comparisons of categorical data were performed with the chi-squared test or Fisher's exact test, as appropriate, while numerical data was analyzed with the Mann–Whitney U test. cTn sensitivity, specificity, and positive and negative predictive value were assessed for T1MI. Differences were considered statistically significant at $p < 0.05$. STATA 14.2 (Stata Corp, College Station, TX) was used for statistical analysis.

Results

Throughout the study period, a total of 8,525 emergency department visits were recorded, during which 2,902 blood tests were conducted. Forty-nine patients residing outside the reference area were excluded from the analysis. Clinicians ordered cTnI testing for 34.7% (n = 989) of the blood samples, with 19.5% (n = 193) yielding positive results. Troponin levels were also measured in the 1,865 (65.3%) excess blood samples from patients for whom cTnI had not been clinically requested (non-clinical troponin), with 10.1% (n = 189) of these testing positive (Fig. 1). In the total analyzed population, cTnI was elevated in 13.4% (382/2,853) of cases.

Patients undergoing clinically indicated troponin testing were older, predominantly male, and had higher rates of hypertension, dyslipidemia, prior myocardial infarction, and chronic pulmonary disease compared to those not tested (Table 1). These patients mainly presented with chest pain or dyspnea and more frequently had repolarization abnormalities on the electrocardiogram. Nearly half of these patients required hospitalization, compared to a lower admission rate (28%) in patients without clinical indication to troponin measurement. A total of 58 T1MI cases were diagnosed, all in the clinical troponin group. Troponin elevations in the non-clinical group corresponded to T2MI in 2.3% and NIMI in 7.8% of patients.

Clinical diagnoses associated with myocardial injury in clinical and non-clinical troponin groups

Approximately 20% of the total population had a diagnosis of cardiovascular disease, more frequently in the clinical troponin group than in the non-clinical group (Table 2).

Prevalence of type 1 myocardial infarction and positive predictive value in patients with clinical troponin and the total series

In the overall cohort, the prevalence of T1MI was 2.0% (95% CI: 1.5–2.5). Among patients with clinically indicated troponin testing, this prevalence increased to 5.8% (95% CI: 4.3–7.2). The positive predictive value for T1MI in the total series as well as in the clinically requested troponin group was 14.8% (95% CI: 13.6–16.2) and 29.5% (95% CI: 26.7–32.4), respectively (Fig. 2). The Influence of clinical characteristics on positive predictive value of elevated cTnI for diagnosis of type 1 myocardial infarction in patients unselected for troponin testing and in patients with clinical decision for troponin testing, are shown in Table 3.

Discussion

This study sheds light on the clinical impact of broadly applying troponin testing protocols in emergency department settings. On the one hand, it highlights that the presence of clinically unsuspected myocardial injury in patients who come to the emergency department for reasons other than suspected acute coronary syndrome is frequent, occurring in approximately 1 in 10 patients,

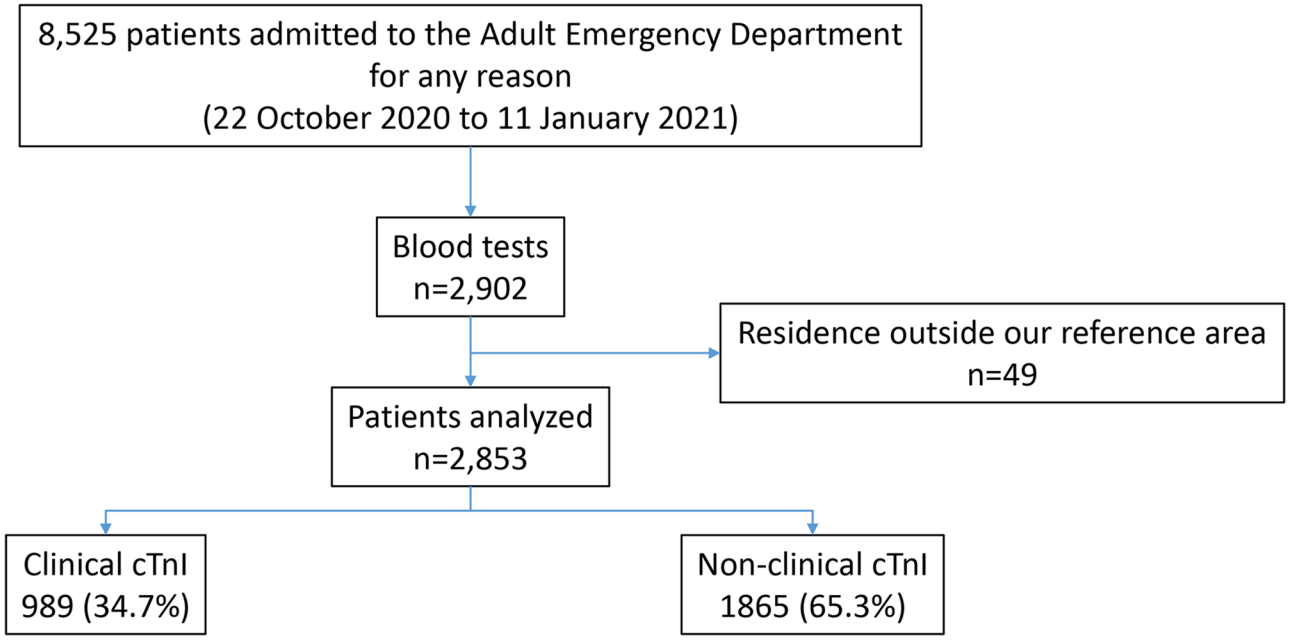

Fig. 1 Flow diagram of patients. The distribution of patients in the two groups of the study is depicted. cTnI: cardiac troponin I

Table 1 Baseline characteristics of patients with and without clinical troponin

	Total (N = 2,853)	Clinical cTnI (N = 989)	Non-clinical cTnI (N = 1,864)	*p*
Demographic Variables				
Age (years)	65.5 (48.5–77.5)	66.5 (52.5–77.5)	64.5 (45.5–77.5)	<0.001
Male sex	1,546 (54.2)	575 (58.1)	971 (52.1)	0.002
Cardiovascular Risk Factors				
Arterial hypertension	1,333 (46.7)	525 (53.1)	808 (43.4)	<0.001
Diabetes mellitus	616 (21.6)	229 (23.2)	387 (20.8)	0.139
Dyslipidemia	930 (32.7)	415 (42.0)	515 (27.7)	<0.001
Smoking	370 (13.0)	120 (12.1)	250 (13.4)	0.333
Cardiovascular History				
Myocardial infarction	203 (7.1)	97 (9.8)	106 (5.7)	<0.001
Heart failure	144 (5.1)	50 (5.1)	94 (5.0)	0.988
Peripheral artery disease	167 (5.9)	63 (6.4)	104 (5.6)	0.392
Cerebrovascular disease	170 (6.0)	53 (5.4)	117 (6.3)	0.324
Chronic kidney disease	290 (10.2)	98 (9.9)	192 (10.3)	0.742
Chronic lung disease	361 (12.7)	181 (18.3)	180 (9.7)	<0.001
Symptoms				
Chest pain	308 (10.8)	297 (30.0)	11 (0.6)	< 0.001
Dyspnea	578 (20.3)	388 (39.2)	190 (10.2)	< 0.001
Other symptoms	2298 (80.6)	683 (69.1)	1615 (86.6)	<0.001
Vital Signs				
Heart rate (bpm)	84 (73–97)	84 (72–100)	83 (73–96)	0.063
Systolic blood pressure (mmHg)	134 (119–151)	135 (120–152)	133 (118–150)	0.166
Oxygen saturation (%)	98 (96–100)	97 (95–99)	99 (97–100)	< 0.001
Electrocardiogram				
Atrial fibrillation	187 (17.4)	71 (11.5)	116 (25.2)	< 0.001
Left or right bundle branch block	126 (11.7)	93 (15.1)	33 (7.2)	< 0.001
ST elevation	45 (4.2)	45 (7.3)	0 (0.0)	< 0.001
ST depression	34 (3.2)	34 (5.5)	0 (0.0)	< 0.001
Negative T wave	65 (6.0)	60 (9.7)	5 (1.1)	< 0.001
Laboratory Tests				
Blood glucose (mg/dL)	112 (97–141)	116 (99–148)	110 (95–137)	<0.001
Glomerular filtration (mL/min per 1.73 m^2)	84 (58–100)	83 (56–96)	85 (59–103)	<0.001
Hemoglobin (g/dL)	13.0 (11.7–14.2)	13.1 (11.7–14.2)	13.0 (11.7–14.2)	0.158
Clinical Evolution				
Hospital admission	1,024 (35.9)	497 (50.3)	527 (28.3)	< 0.001
Coronary angiography	76(2.8)	75(7.6)	1(0.1)	<0.001
In hospital mortality	149(5.2)	71(7.2)	78(4.2)	0.001
Myocardial Injury				
Elevated troponin	382 (13.4)	193 (19.5)	189 (10.1)	<0.001
Type 1 MI (T1MI)	57 (2.0)	57 (5.8)	0 (0.0)	<0.001
Type 2 MI (T2MI)	70 (2.5)	27 (2.7)	43 (2.3)	0.487
Non-ischemic myocardial injury (NIMI)	255 (8.9)	109 (11.0)	146 (7.8)	0.004

The data is represented as numbers (percentages) and medians (interquartile range). cTnI stands for cardiac troponin I

most of whom have T2MI or NIMI. In unselected patients, the prevalence of T1MI was notably low at 2%, with an equally low positive predictive value for elevated cTnI concentrations of 14.9%. Therefore, the diagnostic performance for myocardial infarction with generalized troponin testing without clinical criteria is significantly lower than when troponin is requested based on clinical criteria and even more so when determined explicitly in patients with a high probability of acute coronary syndrome. Considering the widespread use of troponin testing in emergency departments, this information should be regarded as a more significant diagnostic challenge for clinicians managing these patients.

Table 2 Main diagnoses in patients with and without clinical troponin. The diagnoses are grouped into cardiovascular pathologies, major non-cardiovascular pathologies, and other non-cardiovascular pathologies

	Total	Clinical cTnI	Non-clinical cTnI	*p*
	(N = 2,853)	(N = 989)	(N = 1,864)	
Cardiovascular Pathology				
Acute coronary syndrome	62 (2.2)	62 (6.3)	0 (0.0)	<0.001
Chest pain	125 (4.4)	125 (12.6)	0 (0.0)	<0.001
Cerebrovascular disease	98 (3.4)	9 (0.91)	89 (4.8)	<0.001
Heart failure	114 (4.0)	50 (5.1)	64 (3.4)	0.035
Tachyarrhythmia	53 (1.9)	26 (2.6)	27 (1.5)	0.026
Bradyarrhythmia	8 (0.3)	4 (0.4)	4 (0.2)	0.460
Hypertensive crisis	33 (1.2)	15 (1.5)	18 (1.0)	0.190
Syncope	63 (2.2)	24 (2.4)	39 (2.1)	0.563
Pulmonary thromboembolism	21 (0.7)	15 (1.5)	6 (0.3)	<0.001
Cardiorespiratory arrest	15 (0.5)	15 (1.5)	0 (0.0)	<0.001
Non-cardiovascular Pathology				
Anemia	52 (1.8)	10 (1.1)	42 (2.3)	0.018
Respiratory failure	102 (3.6)	42 (4.3)	60 (3.2)	0.159
Renal failure	34 (1.2)	9 (0.9)	25 (1.3)	0.312
Gastrointestinal bleeding	48 (1.7)	3 (0.3)	45 (2.4)	<0.001
Other digestive pathologies	275 (9.6)	9 (0.9)	266 (14.3)	<0.001
Sepsis	22 (0.8)	1 (0.1)	21 (1.1)	0.003
Other infections	220 (7.7)	12 (1.2)	208 (11.2)	<0.001
Neoplasm	30 (1.1)	5 (0.5)	25 (1.3)	0.037
Other Non-cardiovascular Diagnoses				
Other Diagnoses	1,478 (51.8)	553 (55.9)	925 (49.6)	0.001

The data is represented as numbers (percentages). cTnI stands for cardiac troponin I

Troponin testing in emergency departments

Our study is similar to that conducted by Lee et al. [19], who evaluated high-sensitivity cTn concentrations in 918 consecutive patients in an emergency department without suspected acute coronary syndrome, where the treating physician collected blood samples. Elevated troponin was present in 12.4% (114 patients) of the cases, of which 0.2% were classified as T1MI, 0.3% as T2MI, and 11.9% as myocardial injury. Elevated troponin concentrations were associated with older age, impaired renal function, multimorbidity, and adverse physiology. During 912 patient years of follow-up, troponin was a strong predictor of death (HR 1.26 per doubling, 95% CI: 1.06–1.49), independent of age, sex, multimorbidity, and adverse physiology. They concluded that elevated troponin in unselected patients primarily reflects myocardial injury rather than infarction.

On the other hand, Shah et al. examined how patient selection for high-sensitivity cTn testing affects the diagnosis of myocardial infarction in hospitals in the United Kingdom (UK) and the United States (US) [20]. Troponin concentrations were measured in 8,500 patients: 1,054 unselected in the UK and 7,446 selected (5,815 in the UK and 1,631 in the US). Among the unselected UK patients, 13.7% had elevated troponin concentrations, with a positive predictive value of 11.8% for T1MI, data similar to that of our study. In the selected patients, the positive predictive value was 59.7% in the UK and 16.4% in the US. The study concluded that troponin testing without prior clinical evaluation tends to reflect myocardial injury more than infarction, highlighting the importance of proper patient selection to improve diagnostic accuracy.

The diagnostic accuracy of troponin testing is inherently linked to the prevalence of T1MI, which itself is heavily influenced by patient selection practices that differ across healthcare systems [21]. One of the reasons is atypical symptoms in patients with suspected acute coronary syndrome [10, 12]. In addition, differences in the proportion of patients presenting with chest pain among those for whom cTn is requested may reflect differences in the approach to clinical evaluation before testing and other factors influencing physicians' risk perception and, therefore, the need to rule out acute coronary syndrome [16]. In specific emergency departments, particularly in the US, an extraordinary volume of cardiac biomarker requests has been described in patients without any symptoms suggestive of acute coronary syndrome [2, 11]. Our finding that the diagnosis of type 1 myocardial infarction remains unchanged, while diagnoses of type 2 myocardial infarction and non-ischemic myocardial injury increase, is particularly relevant as emergency departments strive to implement rational testing protocols.

Significance of myocardial injury detected in emergency departments: prognostic implications

Although elevated cTn levels without acute coronary syndrome can be challenging to interpret, they provide potentially important clinical information [22, 23]. Many of these patients are recognized by their treating physician as critically ill, although they are not always admitted to the hospital [24]. cTn is a powerful prognostic marker in patients with T2MI or myocardial injury [25, 26]. The detection of myocardial injury may open the opportunity to investigate what possible underlying pathology the heart may have, which could eventually benefit from some differentiated therapeutic action. Still, there are no guidelines on how to investigate these patients, including the role of cardiac monitoring, and no evidence is yet available to suggest that cardiovascular treatments will improve outcomes. More studies are needed to

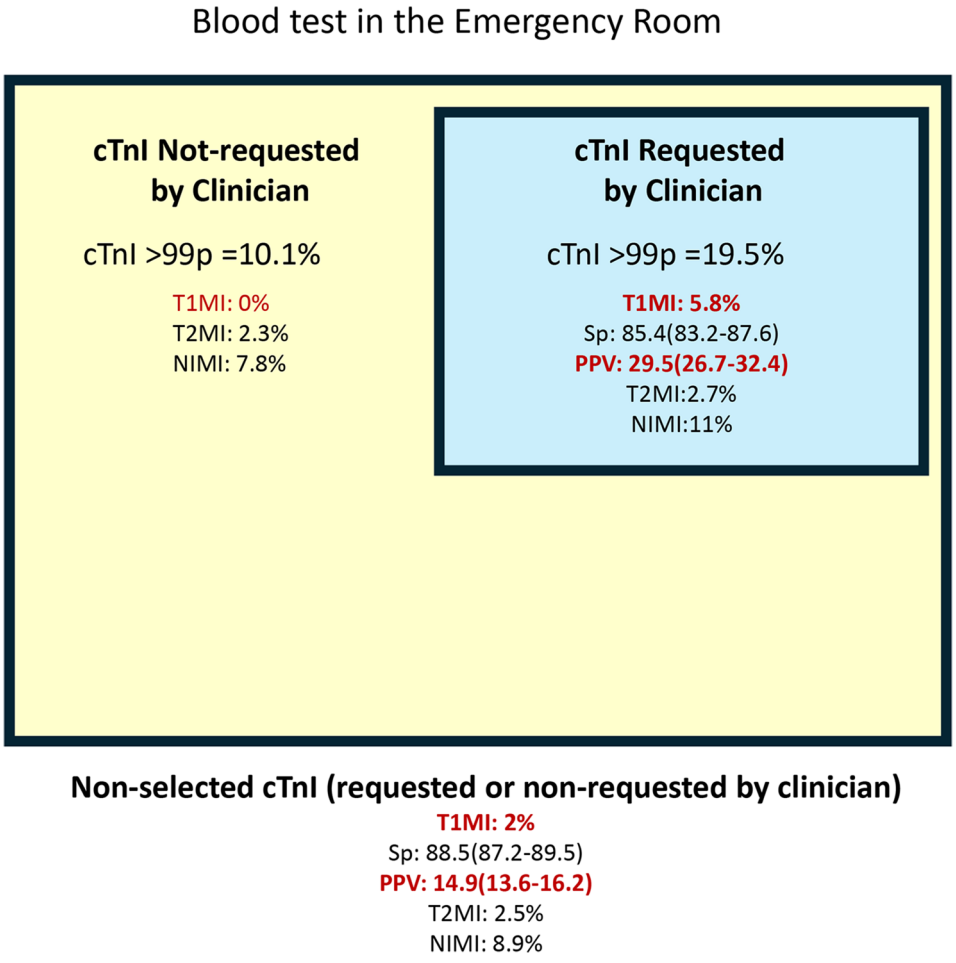

Fig. 2 Prevalence of elevated cardiac troponin I (cTnI) concentrations, type 1 myocardial infarction (T1MI), type 2 myocardial infarction (T2MI), and non-ischemic myocardial injury (NIMI) in unselected patients and those selected for cardiac troponin testing. Sensitivity (Se) and positive predictive value (PPV) of elevated high-sensitivity cardiac troponin for the diagnosis of T1MI in clinically selected patients and in the total population are depicted

systematically evaluate patients with T2MI and myocardial injury, to determine the underlying mechanisms, and to inform the optimal management of these patients.

Strengths

Our study has several strengths, primarily its ability to minimize selection bias by including all consecutive patients who underwent blood sampling in the same emergency department. This allowed us to evaluate diagnostic performance for T1MI in samples where the clinician requested troponin determination and in those where such a request was not made. Second, we used a high-sensitivity analytical method and clear diagnostic criteria to adjudicate T1MI, T2MI, and NIMI, as currently recommended in the Fourth Universal Definition of Myocardial Infarction and used in previous studies [27, 28]. There is only one previous study that has specifically analyzed the diagnostic performance of non-selectively determined troponin in patients treated in the emergency department, and it involves a cohort of 1,054 patients from the United Kingdom [20]. Our series analyzes this phenomenon in a consecutive cohort of 2,853 patients, almost 3 times larger than the British cohort. This allows for greater consistency in the results and greater confidence in the data we present.

Limitations

This study was performed in a single-center setting. Nonetheless, the findings closely align with those reported for selected populations in the UK and unselected cohorts in the US, supporting the generalizability of the results

Conclusions

Broad implementation of high-sensitivity troponin assays without preliminary clinical assessment frequently results in elevated troponin levels. These elevations primarily indicate myocardial injury (type 2 myocardial infarction or non-ischemic injury) rather than type 1 myocardial infarction, thereby significantly reducing

Table 3 Influence of clinical characteristics on positive predictive value of elevated high sensitivity cardiac troponin for diagnosis of type 1 myocardial infarction in patients unselected for troponin testing and in patients with clinical decision for troponin testing

	Stratum	Prevalence (%) (95% CI)	Specificity (%) (95% CI)	Positive Predictive Value (95% CI)
All patients				
Age	≥65	2.5 (1.7–3.3)	82.3 (80.3–84.3)	12.7 (11.0–14.5)
	<65	1.5 (0.9–2.1)	94.4 (93.2–95.6)	21.2 (19.1–23.3)
Chest pain	Yes	15.9 (11.8–20.0)	87.6 (84.0–91.3)	60.5 (55.0–66.0)
	No	0.3 (0.1–0.5)	88.5 (87.2–89.7)	2.7 (2.0–3.3)
ST-segment alterations	Yes	58.1 (46.9–69.4)	48.4 (37.0–59.8)	72.9 (62.8–83.0)
	No	0.5 (0.2–0.8)	88.8 (87.7–90.0)	4.3 (3.6–5.1)
Diabetes	Yes	3.6 (2.1–5.0)	81.8 (78.8–84.9)	16.9 (14.0–19.9)
	No	1.6 (1.1–2.1)	90.2 (88.9–91.4)	13.9 (12.5–15.3)
Previous myocardial infarction	Yes	4.9 (2.0–7.9)	78.2 (72.6–83.9)	19.2 (13.8–24.7)
	No	1.8 (1.3–2.3)	89.1 (87.9–90.3)	14.2 (12.9–15.6)
Chest pain and ST-segment alterations	Yes	62.1 (49.6–74.6)	40.9 (28.3–53.6)	73.5(62.1–84.8)
	No	0.8 (0.4–1.1)	88.8 (87.6–89.9)	6.3 (5.4–7.2)
Chest pain and diabetes	Yes	23.6 (13.8–33.4)	90.9 (84.3–97.6)	77.3 (67.6–87.0)
	No	1.4 (1.0–1.9)	88.3 (87.1–89.5)	11.1 (9.9–12.3)
Chest pain and diabetes peripheral artery disease	Yes	31.8 (12.4–51.3)	80.0 (63.3–96.7)	70.0 (50.9–89.2)
	No	1.8 (1.3–2.3)	88.4 (87.2–89.6)	13.4 (12.2–14.7)
Patients with clinical cTn				
Age	≥65	6.8 (4.6–8.9)	80.2 (76.8–83.6)	26.9 (23.1–30.6)
	<65	4.6 (2.7–6.5)	91.3 (88.7–93.9)	35.6 (31.2–40.0)
Chest pain	Yes	16.5 (12.3–20.7)	87.1 (83.3–90.9)	60.5 (54.9–66.1)
	No	1.2 (0.4–2.0)	84.8 (82.1–87.5)	7.1 (5.2–9.1)
ST-segment alterations	Yes	58.1 (46.9–69.4)	48.4 (37.0–59.8)	72.9 (62.8–83.0)
	No	1.5 (0.7–2.3)	86.7 (84.5–88.9)	10.5 (8.5–12.4)
Diabetes	Yes	9.6 (5.8–13.4)	81.6 (76.6–86.7)	36.7 (30.4–42.9)
	No	4.6 (3.1–6.1)	86.5 (84.1–88.9)	26.3 (23.2–29.5)
Previous myocardial infarction	Yes	10.3 (4.3–16.4)	77.0 (68.6–85.4)	33.3 (24.0–42.7)
	No	5.3 (3.8–6.7)	86.3 (84.0–88.5)	28.8 (25.9–31.8)
Chest pain and ST-segment alterations	Yes	62.1 (49.6–74.6)	40.9 (28.3–53.6)	73.5(62.1–84.8)
	No	2.3 (1.3–3.2)	86.5 (84.3–88.7)	14.6 (12.3–16.9)
Chest pain and diabetes	Yes	23.9 (14.0–33.9)	90.7 (84.0–97.5)	77.3 (67.5–87.0)
	No	4.4 (3.0–5.7)	85.1 (82.8–87.4)	23.4 (20.7–26.1)
Chest pain and diabetes peripheral artery disease	Yes	31.8 (12.4–51.3)	80.0 (63.3–96.7)	70.0 (50.9–89.2)
	No	5.2 (3.8–6.6)	85.5 (83.3–87.7)	27.3 (24.5–30.1)

The data is represented as numbers (percentages). CI stands for confidence interval

the diagnostic accuracy for type 1 myocardial infarction. Therefore, the overall diagnostic performance of high-sensitivity cTn is influenced by patient selection and the presence of comorbid conditions, and physicians should be aware of both aspects when selecting patients for testing and interpreting elevated cTn concentrations in their practice. In order to increase the diagnostic performance of troponin testing in the emergency department, it is necessary to better define the clinical profile of the patient who has a probability of acute coronary syndrome, not only based on the characteristics of chest pain, but also on data from their cardiovascular history and risk factors.

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Author contributions
Conceptualization, A.B.; methodology, A.B., O.M.P.; software, A.B.; validation, A.B., O.M.P.; formal analysis, A.B., O.M.P.; investigation, M.L.G., J.R.D., M.R., M.G., I.F.; resources, A.B.; data curation, A.B., O.M.P., A.C.; writing—original draft preparation, A.B.; writing—review and editing, A.B., O.M.P., A.C.; visualization, A.B.; supervision, A.B.; project administration, A.B. All the authors have read and agreed to the published version of the manuscript.

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Data availability
The data used and/or analyzed in this study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
The study was approved by our Institutional Review Board (Comité Ético de Investigación con Medicamentos del Institut d'Investigació Sanitària Pere Virgili) and conducted in accordance with the Declaration of Helsinki. The approval registry number is CEIM 195/2020. Since the leftover material was acellular, the ethics committee determined that individual patient consent was not required.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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