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Superior early diagnostic performance of a sensitive cardiac troponin assay as compared to a standard troponin test in the diagnosis of acute myocardial infarction

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Abstract

Background: New generation cardiac troponin assays have sufficient precision to detect and quantify plasma troponin concentrations below the lower threshold of detection of the currently employed troponin tests. However, diagnostic performance of the newer generation assays in daily clinical practice is not well established.

Aim: To evaluate the diagnostic performance of a sensitive assay as compared to a standard assay in a single reading at admission in the diagnosis of acute myocardial infarction (AMI) in patients presenting to the Emergency Department with chest pain.

Methods: The study comprised 187 consecutive patients admitted to the Institute of Cardiology in Warsaw in June and July 2010 with chest pain in whom the attending physician ordered troponin assay to rule AMI in or out. In all of these patients, in addition to the standard Dimension Flex Troponin I (Siemens Healthcare Diagnostics, Inc.) the sensitive Architect Stat Troponin I (Abbott Diagnostics) test was assayed. The triage of patients as well as all diagnostic and treatment decisions were left to the discretion of the attending physician who was blinded to the sensitive troponin test readings. The final diagnosis was adjudicated by a team of two cardiologists on the basis of all the available medical records except for sensitive troponin test results.

Results: Mean age of the study cohort (n = 187) was 64.3 ± 13.9 years and 119 (63.6%) were males. The final diagnosis of AMI was adjudicated in 84 (44.9%) patients (mean age 67.5 ± 12.9 years; 119 [63.6%] males). Receiver operating characteristic (ROC) analysis showed greater area under the curve (AUC) for the sensitive cardiac troponin assay compared to the standard assay (AUC = 0.916, 95% CI = 0.866–0.951 *vs* AUC = 0.863, 95% CI = 0.806–0.909, respectively; p = 0.02) in a single reading at admission. Sensitive assay was characterised by higher sensitivity (87%), specificity (88%), positive (86%) and negative (89%) predictive values in the detection of AMI compared to the standard troponin test (82%, 81%, 78%, and 85% respectively).

Conclusions: The newer generation sensitive cardiac troponin assay presented superior diagnostic accuracy in the diagnosis of AMI compared to the standard troponin test in a single reading at admission with improved sensitivity and specificity. The sensitive troponin assay has the potential to improve early detection and/or exclusion of AMI.

Key words: sensitive cardiac troponin assay, acute myocardial infarction, laboratory diagnostics

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INTRODUCTION

Acute myocardial infarction (AMI) remains one of the leading causes of mortality and morbidity in industrialised countries [1–3]. Treatment options are now in place that are capable of limiting, or even preventing, early and late adverse consequences of AMI [4, 5]. However, early and accurate diagnosis of AMI enables timely and appropriate implementation of these therapies, and thus is key to their effectiveness. In the very early stages of ST-elevation myocardial infarction (STEMI) clinical signs and symptoms coupled with ECG changes are often sufficient to make appropriate diagnosis and initiate treatment without unnecessary delay caused by waiting for elevation of troponin levels [4, 6, 7]. Nevertheless, early and accurate diagnosis of non-ST elevation myocardial infarction (NSTEMI) remains a daily clinical challenge. On the other hand, fast, accurate, and reliable exclusion of the ongoing myocardial necrosis in the environment of the Emergency Department (ED) is crucial to ensure appropriate triage of patients, prevent EDs' overcrowding, and reduce the overall cost burden of healthcare [8].

Cardiac troponins are structural proteins specific to cardiomyocytes and as such play a central role in the diagnostic process of AMI [9, 10]. The significance of troponin blood concentrations has recently been corroborated by The Joint ESC/ACCF/AHA/WHF Task Force for the Universal Definition of Myocardial Infarction. The Task Force states that in order to diagnose AMI a rising and/or falling pattern of troponin blood concentration with at least one value above the 99th percentile of the upper reference limit of the healthy reference population needs to be detected [11]. Elevated plasma cardiac troponin levels should be interpreted together with the entire clinical context and AMI may be considered if angina, ischaemic ECG abnormalities, or newly present myocardial dysfunction are detected.

A substantial time gap exists between the onset of MI and the rise of cardiac troponin blood levels detectable by the current troponin assays with sufficient accuracy. This delay often requires subsequent troponin tests at six-hourly intervals to confirm or exclude the diagnosis of AMI [11]. Recent developments in immunoassay technology have allowed for the construction of new generation troponin assays capable of detecting much lower concentrations of cardiac troponins in the blood with high precision [10, 12]. These assays may allow for the detection of raised troponin with greater accuracy and earlier in the process of the ongoing myocardial necrosis. However, diagnostic performance of these new generation assays in daily clinical practice is not well established. Therefore, the aim of the current analysis was to evaluate the diagnostic performance of the sensitive troponin assay in comparison with the standard troponin assay in the diagnosis of AMI in a single reading at admission in chest pain patients.

METHODS

Study population

The study comprised consecutive patients admitted to the Institute of Cardiology with chest pain in whom the attending physician suspected acute coronary syndrome (ACS) and ordered troponin assay. The total number of 187 patients were enrolled from June to July of 2010.

Study methods

The study was observational in nature, therefore the triage of patients as well as all the diagnostic and treatment decisions were left to the discretion of the attending physician who remained blinded to the sensitive troponin assay readings.

Blood samples for troponin assays were collected within 24 hours from the onset or peak of symptoms. The routine troponin test was Dimension Flex Troponin I (Siemens Healthcare Diagnostics, Inc.) and this assay was used in the current analysis as the reference. Additionally, in all of the study patients, sensitive Architect Troponin I (Abbott Diagnostics) was assayed as part of its validation at the hospital's laboratory. Blood samples for the determination of investigational cardiac troponin assay were stored frozen at –20°C until they were assayed in a blinded fashion. The remaining laboratory tests were performed as ordered by the attending physician. All laboratory tests were performed in the Diagnostyka Laboratory sp. z o.o. SK in the Institute of Cardiology.

The final diagnosis was adjudicated by a team of two experienced cardiologists on the basis of all the available medical records with the exception of sensitive troponin test results. If there was disagreement between the two cardiologists, the opinion of a third, independent, specialist was sought. The final diagnosis fell into one of three predefined categories: (1) AMI (STEMI or NSTEMI); (2) unstable angina (UA); or (3) non-acute coronary syndrome (non-ACS). The diagnosis of AMI was established according to the criteria proposed by The Joint ESC/ACCF/AHA/WHF Task Force for the Universal Definition of Myocardial Infarction, i.e. detection of rise and/or fall of cardiac biomarkers of necrosis (troponin) with at least one value above the 99th percentile of the upper reference limit (URL) together with myocardial ischaemia as evidenced by typical angina, ischaemic ECG changes, imaging evidence of new loss of viable myocardium or new regional wall motion abnormalities [11]. Unstable angina was diagnosed in patients in whom coronary artery disease was established based on previously or currently performed invasive or noninvasive coronary angiography or who had documented exertion ischaemia on exercise test, and who had typical rest angina or recent deterioration of exertion angina without elevation of blood troponin levels. Patients in the third category had chest pain with apparent cardiac cause other than ACS (e.g. perimyocarditis, tachyarrythmias) or any other non-cardiac cause (e.g. acute pulmonary embolism) or had

chest pain of unclear origin excluding ACS. A subgroup of patients with chest pain of apparent origin other then ACS and troponin elevation above the 99th percentile was prespecified.

The adjudication process was based on the Dimension Flex Troponin I assay (Siemens Healthcare Diagnostics, Inc., Newark, DE, USA) with the lower limit of detection (LOD) equal to 0.04 ng/mL, 99th percentile of URL at 0.07 ng/mL with imprecision at this concentration equal to 15–22% coefficient of variation (CV), and imprecision of 10% at the concentration of 0.14 ng/mL, as specified by the manufacturer. The sensitive troponin assay tested in the current analysis was Architect Stat Troponin I assay performed with the use of the Architect system (Abbott Diagnostics, Abbott Park, IL, USA), with LOD equal to 0.01 ng/mL, 99th percentile of URL at 0.028 ng/mL with imprecision at this concentration equal to 15% CV, and a CV of less than 10% at 0.032 ng/mL, as specified by the manufacturer.

The study was approved by the local ethics committee and conformed to the principles of the Declaration of Helsinki. The sensitive troponin assay was provided by the manufacturer free of charge. The study design, data gathering and analysis, as well as drafting of the manuscript and its subsequent submission for publication have been carried out by the authors independently of and without any interference on the part of the manufacturer.

Statistical analysis

The data analysis was performed using SPSS statistical analysis software (SPSS Inc., Chicago, IL, USA) and Medcalc statistical software (Medcalc, Belgium). Continuous variables were presented as mean ± SD and compared using Mann-Whitney U test. Categorical variables were summarised as numbers and percentages and compared using χ^2 or Fishers' exact test as appropriate. Receiver operating characteristic (ROC) curves were constructed to obtain areas under the curves (AUC), sensitivity, specificity, as well as positive and negative predictive values (NPV and PPV, respectively) of cardiac troponin levels determined at presentation in the diagnosis of AMI. The comparison of areas under the ROC curves (AUC) was performed as recommended by DeLong et al. [13]. Two-tailed hypotheses testing was used in all analyses and a p-value of less than 0.05 was considered statistically significant.

RESULTS

Baseline data

The study cohort comprised 187 patients of whom 119 (63.6%) were male and the mean age was 64.3 ± 13.9 years. The AMI was the adjudicated diagnosis in 84 (44.9%) patients. A total of 43 (23.0%) patients had STEMI and 41 (21.9%) had NSTEMI. In the remaining 103 (55.1%) cases: UA was the final adjudicated diagnosis in 11 (5.9%) and non-ACS in 92 (49.2%) patients. Demographic and clinical characteristics of the study group are presented in Table 1.

Troponin levels

Patients with AMI had higher troponin concentrations at admission compared to patients in whom AMI was excluded (11.723 ± 36.263 *vs* 0.220 ± 1.177 ng/mL, respectively for the sensitive troponin assay, and 11.32 ± 32.80 *vs* 0.29 ± ± 1.27 ng/mL, respectively for the standard troponin assay; $p < 0.01$ for both). Patients with UA and non-ACS had similar troponin levels as determined by sensitive (0.007 ± 0.004 *vs* 0.245 ± 1.244 ng/mL; p = NS) or standard (0.04 ± 0.03

Table 1. Selected demographic and clinical characteristics of the study group

Variable	Overall study group (n = 187)	Patients with AMI (n = 84)	Patients without AMI (n = 103)	P
Male gender	119 (63.6%)	55 (65.5%)	64 (62.1%)	NS
Age	64.3 ± 13.9	67.5 ± 12.9	61.7 ± 14.4	< 0.01
Age ≥ 65 years	94 (50.3%)	46 (54.8%)	48 (46.6%)	NS
Hypertension	114 (61.0%)	58 (69%)	56 (54.4%)	0.05
Hyperlipidaemia	68 (36.4%)	49 (58%)	19 (18.4%)	< 0.001
Diabetes mellitus	27 (14.4%)	17 (20.2%)	10 (9.7%)	0.06
Smoking history	26 (13.9%)	18 (21.4%)	8 (7.8%)	0.01
Family history	7 (3.7%)	5 (6.0%)	2 (1.9%)	NS
Previous MI	33 (17.6%)	14 (16.7%)	19 (18.4%)	NS
Previous PTCA	30 (16.0%)	11 (13.1%)	19 (18.4%)	NS
Previous CABG	15 (8.0%)	6 (7.1%)	9 (8.7%)	NS
History of stroke	3 (1.6%)	2 (2.4%)	1 (1.0%)	NS

Data is presented as the number of patients (percentage) or as mean ± SD; a p value refers to the comparison of values between patients with and without AMI; AMI — acute myocardial infarction; CABG — coronary artery bypass grafting; MI — myocardial infarction; PTCA — percutaneous coronary intervention

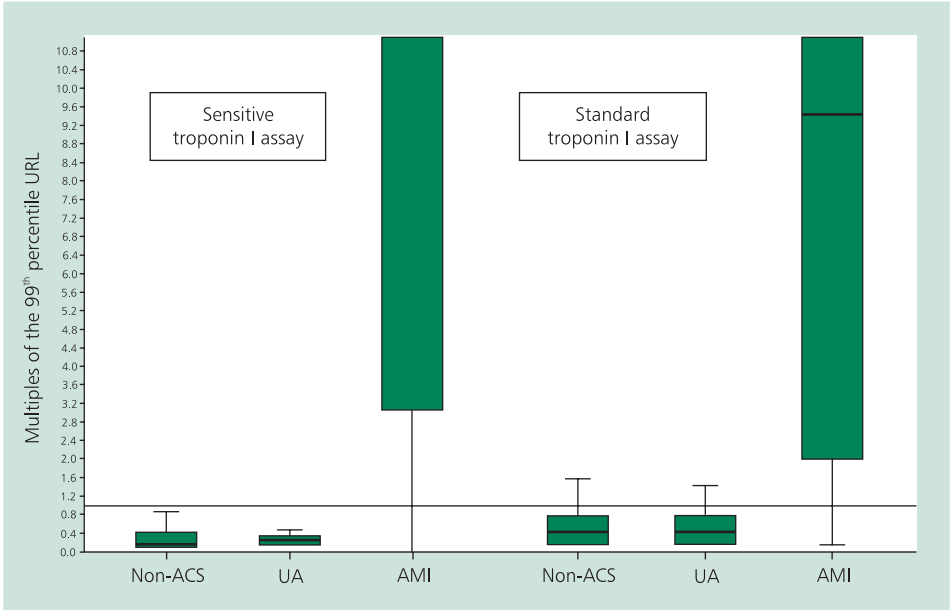

Figure 1. Cardiac troponin I levels on admission of patients with chest pain determined by the two studied troponin assays. Troponin levels at presentation of patients with chest pain admitted to the Institute of Cardiology are demonstrated as multiples of the 99th percentile specific to each of the two tests. The boxes represent interquartile ranges, the horizontal line in each box represents the median (the absence of a horizontal line indicates a median > 10 times the 99th percentile); the whiskers represent the minimum and maximum values with exclusion of outliers that were more than 1.5 times of interquartile range. Multiples of the 99th percentile greater than 10 are not shown; AMI — acute myocardial infarction; non-ACS — non-acute coronary syndrome; UA — unstable angina; URL — upper reference limit

vs 0.32 ± 1.35 ng/mL; p = NS) assay. Troponin levels determined at presentation in subgroups of patients categorised to the three diagnostic groups, i.e. AMI, UA, and non-ACS are presented in Figure 1 for both sensitive (panel A) and standard (panel B) troponin assays.

Diagnostic accuracy of the studied troponin assays

The ROC analysis showed greater AUC for the sensitive troponin assay compared to the standard troponin assay (AUC 0.916, 95% CI 0.866–0.951 *vs* AUC 0.863, 95% CI 0.806––0.909, respectively; p = 0.02) (Fig. 2). At the cut-off point at the 99th percentile of the URL specific to each of the two troponin tests sensitivity, specificity, PPV and NPV for AMI diagnosis amounted to 87%, 88%, 86%, and 89% respectively for sensitive assay and 82%, 81%, 78%, 85% respectively for the standard assay (Table 2). Diagnostic parameters of the two analysed troponin assays for the cut-off points at 99th percentile URL and also at LOD and concentration at which the assay reaches 10% CV are all shown in Table 2.

The ROC analysis of the diagnostic performance of sensitive troponin assay in a single reading at admission yielded similar results across subpopulations of patients with STEMI (AUC 0.906, 95% CI 0.847–0.948) and NSTEMI (AUC 0.926, 95% CI 0.871–0.963), older (≥ 65 years) (AUC 0.910, 95% CI 0.833–0.959) and younger (< 65 years) (AUC 0.918, 95% CI 0.842–0.965) patients, and also in men (AUC 0.895, 95% CI 0.826–0.944) and women (AUC 0.949, 95% CI 0.866–

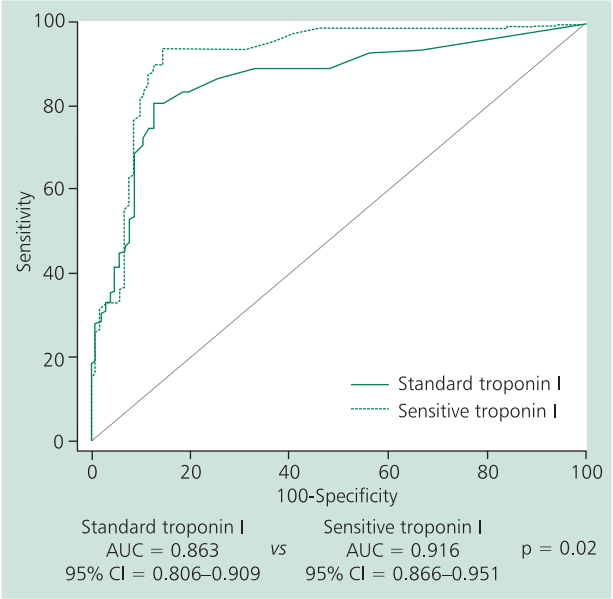

Figure 2. Receiver-operating characteristic curves (ROC) depicting diagnostic performance of the studied troponin assays in the diagnosis of acute myocardial infarction in a single reading at admission. The continuous green line represents diagnostic performance of the standard troponin I, and the dashed green line represents diagnostic performance of the sensitive troponin I. The figure incorporates areas under the curve (AUC) together with 95% confidence intervals (CI) for both assays and the p value for the difference between them

Table 2. Diagnostic parameters of the two studied troponin I assays in a single reading at admission of patients with chest pain

	Sensitivity	Specificity	PPV	NPV
Sensitive troponin I assay				
LOD (0.01 ng/mL)	94%	77%	77%	94%
99th percentile (0.028 ng/mL)	87%	88%	86%	89%
10% CV (0.032 ng/mL)	86%	89%	87%	88%
Standard troponin I assay				
LOD (0.04 ng/mL)	89%	67%	69%	88%
99th percentile (0.07 ng/mL)	82%	81%	78%	85%
10% CV (0.14 ng/mL)	74%	88%	84%	80%

CV — coefficient of variation; LOD — lower limit of detection; NPV — negative predictive value; PPV — positive predictive value

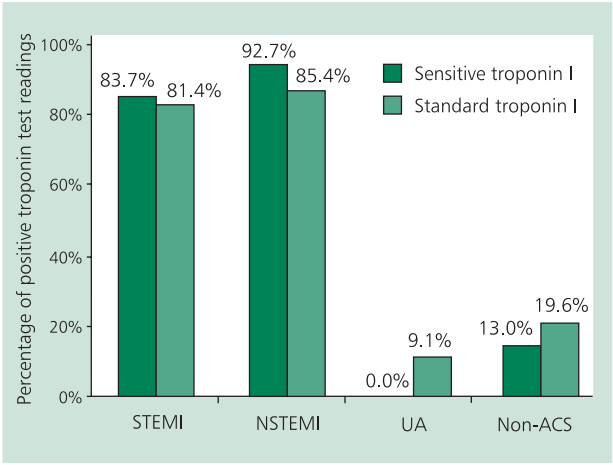

Figure 3. Column chart presenting percentage of patients with positive admission troponin test readings across subgroups of patients with final diagnosis of ST elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), unstable angina (UA), and non-acute coronary syndrome (non-ACS)

–0.988). The percentage of patients with positive admission troponin test readings across subgroups of patients with final diagnoses of STEMI, NSTEMI, UA, and non-ACS is presented in Figure 3.

In patients in whom AMI was excluded but who had troponin elevation above the 99th percentile as determined by standard assay at admission (n = 19, 13 [68%] male) the excessive troponin release was attributable to decompensated heart failure (n = 1), cardiac arrhythmia (n = 4), myocarditis (n = 2), acute pulmonary embolism (n = 3), and dissecting aneurysm of the ascending aorta (n = 1), and cardiac contusion in an accident (n = 1); in seven patients, no apparent cause of troponin elevation was evidenced. With regard to the sensitive assay, in patients in whom AMI was excluded but who had troponin elevation above the 99th percentile as determined by sensitive assay at admission (n = 12, 8 [67%] male), the excessive troponin release was attributable to decompensated heart failure (n = 2), cardiac arrhythmia (n = 4), myocarditis (n = 2), acute pulmonary embolism (n = 2), and dissecting aneurysm of the ascending aorta (n = 1); in one patient no apparent cause of troponin elevation was evidenced.

DISCUSSION

Main study findings

The results of the current analysis show that a sensitive troponin assay compared to a standard troponin test presents superior diagnostic performance in the diagnosis of AMI in a single reading at admission. The superior diagnostic performance was evidenced by greater area under the ROC curve for the sensitive assay and pertained with regard to both sensitivity and specificity at the 99th percentile URL cut-off point. The diagnostic accuracy of the sensitive assay proved to be consistent among subgroups of patients with STEMI and NSTEMI, men and women, as well as older and younger patients.

Troponin assays and AMI guidelines

International guidelines on the New Definition of Myocardial Infarction state that to make the diagnosis of AMI a rise and/or fall of cardiac biomarkers of necrosis (preferably troponin) with at least one value above the 99th percentile of URL should be detected [11]. Troponin elevation is to be interpreted in the clinical settings of the ongoing myocardial ischaemia.

This renders troponin a key element in the work-up of patients with suspected AMI, especially with regard to NSTEMI. From the clinical standpoint, the treatment of STEMI should be initiated based on clinical presentation and ECG abnormalities even if troponins have not yet been released into circulation in sufficiently high quantities [4, 7]. The guidelines also specify that troponin assays be characterised by precision (CV) at the 99th percentile URL equal to or less than 10%. This requirement could not have been fulfilled in the past due to technical shortcomings of contemporary tests. Imprecision greater than 10% may lead to both false positive and

false negative results, especially in patients with blood troponin levels close to the 99th percentile URL. The new generation, sensitive assays are capable of detecting lower concentrations of cardiac troponins with increased precision at the 99th percentile of URL [10, 12]. Sensitive troponin assays have been designed to detect myocardial necrosis earlier from its onset with sufficiently high diagnostic precision.

The sensitive troponin assay

The sensitive assay used in the current study has imprecision at the 99th percentile of URL equal to 15%, which is close to the recommended level of 10% and outperforms the reference test (CV 15–22%). It also detects much lower levels of troponin, although precise comparisons of troponin levels obtained with various tests is precluded by their lack of standardisation. Indeed, in our study, improved analytical parameters of the sensitive assay translated into improved clinical diagnostic performance.

Sensitive tests are characterised by lower troponin concentration at the 99th percentile URL and are capable of detecting very low levels of troponins, often also in patients with stable coronary artery disease or even apparently healthy individuals [10, 14]. This is why some authors argue that the introduction of sensitive assays to daily clinical practice may increase the frequency of NSTEMI diagnosis due to a decrease in LOD and 99th percentile URL [15, 16]. Although it may hold true for other tests or clinical circumstances our data proves otherwise. As determined at admission, the sensitive assay showed fewer false positive results. So not only was specificity retained but it actually improved with the sensitive test. This fact most probably results from greater CV of the sensitive test compared to standard assay at low blood troponin concentrations (i.e. close to the 99th percentile). By definition, in lower ranges of protein concentration a standard test will more often show false positive (as well as false negative) results only due to its insufficient laboratory precision.

In support of this assertion, out of 12 false positive results of the sensitive test all but one (8.3%) had a cause of troponin elevation attributable to specific causes other than AMI, whereas in seven out of 19 (36.8%) false positive standard test results, no apparent attributable cause was found. It may be speculated that in patients in whom no apparent attributable cause of troponin elevation was found, the cause was the laboratory imprecision of the assay.

It has to be stressed that the sensitive tests do not merely shift the 99th percentile to lower levels but rather re-establish it based on improved lower limit of detection and coefficient of variation (less imprecision). Importantly, the presence of myocardial injury, i.e. troponin elevation due to causes other than acute coronary ischaemia, will still require careful clinical judgment to be distinguished from AMI [17–19]. Lack of a clinical tool that is capable of distinguishing myocardial injury from myocardial necrosis with 100% sensitivity and specificity is a limitation of the current study and of other clinical research in this area. It also constitutes a challenge for contemporary acute cardiac care, especially in the case of NSTEMI. The greater prevalence of myocardial injury among men caused a slight difference between AUC for men and women in our cohort.

It is worth noting that the scope of troponin concentrations below the 99th percentile of URL in patients without AMI has been shown to portend strong prognostic information regarding cardiovascular and overall morbidity and mortality [14, 20–23]. Thus, sensitive troponin assays may open a whole new chapter in preventive medicine and may broaden our diagnostic and prognostic arsenal.

Perspective

Although our report, as well as others dealing with this subject suggest that sensitive assays may improve early diagnosis of AMI [24–26], a few issues remain unresolved. First, there is no data demonstrating whether introduction of the new generation assays would shorten the time from symptoms onset to treatment initiation and ultimately improve patients' outcomes. Second, studies with serial troponin testing using sensitive assays need to be performed. It has been suggested that sensitive tests may reliably exclude ongoing myocardial necrosis with serial testing performed at three-hourly instead of six-hourly intervals [27]. If this is proved, it may bring some improvement to healthcare in general and especially to Emergency Departments in terms of cost-savings and overcrowding [8].

CONCLUSIONS

The results of the current analysis indicate that a sensitive troponin assay determined at admission compared to a standard troponin test is characterised by superior diagnostic performance with improved sensitivity and specificity in ruling AMI in or out. The diagnostic accuracy of the sensitive assay proved to be consistent among subgroups of patients with STEMI and NSTEMI, men and women, older and younger patients.

Conflicts of interest: *Radosław Pracoń — a research grant from Abbott Diagnostics; Mariusz Kruk — a research grant from Abbott Diagnostics; Barbara Jakubczak — none declared; Marcin Demkow — none declared; Zofia T. Bilińska — none declared*

The sensitive troponin assay was provided by the manufacturer free of charge. The study design, data gathering and analysis, as well as drafting of the manuscript and its subsequent submission for publication have been carried out by the authors independently of and without any interference on the part of the manufacturer.

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Wyższa wartość diagnostyczna czułego testu na troponinę w porównaniu z testem standardowym w diagnostyce ostrego zawału serca

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Streszczenie

Wstęp: Wczesne rozpoznanie ostrego zawału serca (AMI) warunkuje wdrożenie skutecznej terapii. Troponiny (Tn) sercowe pełnią zasadniczą rolę w postępowaniu diagnostycznym. Jedną z istotniejszych wad tego markera jest opóźnienie czasowe między początkiem martwicy miokardium a wykrywalnym wzrostem stężenia Tn w surowicy krwi. Postęp technologiczny umożliwił w ostatnim czasie stworzenie tzw. czułych testów na Tn wykrywających znacznie niższe niż dotychczas stężenia tego białka w surowicy krwi przy zachowaniu wysokiej precyzji diagnostycznej. Wartość diagnostyczna czułych testów w codziennej praktyce klinicznej nie jest jednak wystarczająco dobrze udokumentowana.

Cel: Celem niniejszej pracy była ocena wartości diagnostycznej pierwszego oznaczenia stężenia Tn za pomocą czułego testu w porównaniu z testem standardowym w diagnostyce AMI.

Metody: Do badania włączono kolejnych 187 pacjentów przyjętych do Instytutu Kardiologii z bólem w klatce piersiowej, u których lekarz prowadzący podejrzewał ostry zespół wieńcowy (OZW) i zlecił oznaczenie stężenia Tn w surowicy krwi. Badanie miało charakter obserwacyjny. Wszystkie kliniczne decyzje dotyczące diagnostyki i postępowania terapeutycznego pozostawiono lekarzowi prowadzącemu. Testem referencyjnym (standardowym) był test Dimension Flex Troponin I (Siemens Healthcare Diagnostics, Inc.). Dodatkowo z pierwszej próbki krwi pobranej w celu oznaczenia Tn standardowej oznaczono stężenie Tn czułym testem Architect Stat Troponin I (Abbott Diagnostics). Lekarz prowadzący nie miał wglądu w wynik stężenia Tn oznaczonej czułym testem. Ostateczną diagnozę stawiało 2 niezależnych kardiologów na podstawie dostępnych danych klinicznych i wyników badań dodatkowych uwzględniających wartości Tn oznaczonych testem standardowym. Rozpoznanie zostało przyporządkowane do jednej z trzech kategorii: (1) AMI, (2) niestabilna choroba wieńcowa, (3) ból w klatce piersiowej o etiologii innej niż OZW. Diagnozę AMI oparto na standardowych kryteriach (*the Universal Definition of Myocardial Infarction*). Niestabilną chorobę wieńcową rozpoznano u pacjentów, którzy zgłaszali typowy ból wieńcowy spoczynkowy lub jego nasilenie w ostatnim czasie bez wzrostu stężenia Tn we krwi, i którzy mieli udokumentowaną obecnie lub uprzednio chorobę wieńcową na podstawie badania koronarograficznego lub za pomocą prób obciążeniowych.

Wyniki: W analizowanej grupie obejmującej 187 pacjentów 63,6% (n = 119) stanowili mężczyźni, średni wiek wyniósł 64,3 ± ± 13,9 roku. Ostateczne diagnozę AMI postawiono u 84 (44,9%) pacjentów, u 43 (23%) rozpoznano zawał z uniesieniem odcinka ST (STEMI), a u 41 (21,9%) zawał bez uniesienia odcinka ST (NSTEMI). Niestabilną chorobę wieńcową stwierdzono u 11 (5,9%) osób, a ból w klatce piersiowej o etiologii innej niż OZW u pozostałych 92 (49,2%) pacjentów. Analiza ROC dla stężenia Tn w surowicy krwi w pojedynczym oznaczeniu przy przyjęciu w diagnostyce AMI wykazała istotnie większe pole pod krzywą (AUC) dla testu czułego w porównaniu z testem standardowym (odpowiednio AUC 0,916; 95% CI 0,866–0,951 v. AUC 0,863; 95% CI 0,806–0,909; p = 0,02). Dla punktu odcięcia równego 99-percentylowi górnej granicy referencji stężenia Tn w zdrowej populacji dorosłych osób czułość, swoistość, dodatnia i ujemna wartość predykcyjna wyniosły odpowiednio 87%, 88%, 86% i 89% dla testu czułego oraz 82%, 81%, 78% i 85% dla testu standardowego; AUC dla czułego testu na Tn było podobne w populacji pacjentów ze STEMI i NSTEMI, osób starszych (≥ 65. rż.) i młodszych (< 65. rż.) oraz u mężczyzn i kobiet. W przypadku wyników fałszywie dodatnich dla testu standardowego (n = 19) oraz dla testu czułego (n = 12) odpowiednio w 12 (63,2%) i w 11 (97,1%) przypadkach występowała klinicznie jawna przyczyna wzrostu Tn inna niż AMI.

Wnioski: Czuły test wykazuje wyższą wartość diagnostyczną w porównaniu z testem standardowym w diagnostyce AMI u chorych z bólem w klatce piersiowej w pojedynczym oznaczeniu przy przyjęciu. Dla punktu odcięcia równego 99. percentlowi górnej granicy referencyjnej stężenia Tn w zdrowej populacji test czuły wykazuje zarówno wyższą czułość, jak i zwiększoną swoistość. Wprowadzenie testu czułego do praktyki klinicznej może usprawnić wczesne rozpoznanie i/lub wykluczenie AMI.

Słowa kluczowe: czuły test na troponinę sercową, ostry zawał serca, diagnostyka laboratoryjna

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