Impact of Point-of-care Testing in the Emergency Department Evaluation and Treatment of Patients with Suspected Acute Coronary Syndromes

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Abstract

Objectives: To assess the impact of point-of-care testing (POCT) for troponin I (cTnI) measurement on the time to anti-ischemic therapy (TAIT) for patients with suspected non–ST-segment elevation acute coronary syndrome (NSTE-ACS) presenting to the emergency department (ED).

Methods: This was an open-label, randomized, single-center trial conducted in a university-affiliated hospital. cTnI measurement of patients with suspicion of NSTE-ACS coming to the ED was randomly allocated to POCT or central hospital laboratory testing (CHLT). The authors compared patients' baseline characteristics, time to anti-ischemic therapy, and medical outcomes between the randomized groups, in all study participants and in high-risk NSTE-ACS (cTnI level ≥ 0.10 μg/mL), and in those with low suspicion ACS (no chest pain and no ST deviation).

Results: Of the 860 patients enrolled, 113 were high-risk NSTE-ACS patients, including 53 (46.9%) allocated to POCT and 60 (53.1%) to CHLT. POCT was associated with decreased time to anti-ischemic therapy of about three-quarters of an hour, which was due to a shorter time to physician notification of cTnI level, in both all and subgroup participants. In contrast, neither ED length of stay nor medical outcomes differed between study groups.

Conclusions: Point-of-care testing for cTnI measurement might be clinically relevant for ED patients with a suspicion of NSTE-ACS, particularly for high-risk patients with a low suspicion of ACS.

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Rapid diagnosis of patients with high-risk non-ST elevation acute coronary syndromes (NSTE-ACSs) may be critical, as recent findings suggest that adverse outcomes increase with increasing time to coronary angiography and related treatment.[1–3] The key components of risk stratification are patient symptoms,

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The corresponding author certifies, on behalf of all authors, that Dade Behring had no role, either directly or through a third party, in gathering or preparation of data or in the writing of the manuscript.

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physical and electrocardiogram (ECG) findings, and biomarkers of cardiac injury.[4] Because many patients with ACS in the emergency department (ED) are ''low suspicion'' (i.e., have neither chest pain nor a diagnostic ECG, as defined by high-risk findings such as ischemic ST-segment elevation, depression, T-wave inversion), biomarkers can help physicians to identify ACS and to stratify risk.[5–9] Because biomarkers are assessed in a central hospital laboratory outside the ED, they are often the time-limiting step in diagnosis of ACS.[4,8,10,11]

Cardiac-specific troponins reliably allow early identification of patients with acute myocardial infarction and are of critical prognostic value, especially troponin I (cTnI).[12] Moreover, the diagnosis of acute myocardial infarction depends on troponin testing in up to 50% of patients when the ECG is nondiagnostic.[12,13] For the purposes of this report, we define patients as ''high risk'' if they have an elevated troponin; these patients must receive timely anti-ischemic therapy and are candidates for early invasive care.[3,14–20]

Point-of-care testing (POCT) has become popular to hasten the availability of laboratory test results.[21] Faster results are of special interest in the ED to reduce the time to treatment.[22–24] However, the impact of POCT on daily practice is poorly documented, particularly for ED patients with suspicion of NSTE-ACS.[25] We hypothesized that POCT for cTnI measurement in the ED would hasten care, particularly for patients without chest pain or a diagnostic ECG. Our study aim was to compare time to anti-ischemic treatment (TAIT) and medical outcomes for patients with a suspicion of NSTE-ACS by place of cTnI testing: POCT at the ED versus central hospital laboratory testing (CHLT).

METHODS

Study Design

We conducted an open-label, randomized controlled trial of adults with suspicion of ACS presenting to our ED. Patients enrolled in this study were randomly allocated to either POCT or CHLT for measuring cTnI level. Blood samples were the unit of randomization. Randomization in blocks of 20 was done with computer generated codes prior to start of the study by our hospital clinical research unit (PM, PC), which was not involved in data collection or patient care. Allocations were concealed in consecutively numbered, sealed, opaque envelopes, safely stocked in the ED. The institution's review board for the protection of human subjects approved the study protocol and patient informed consent procedures. The trial is registered under number ISRCTN92275378 (http://www.controlled-trials.com/ISRCTN92275378).

Study Setting and Population

With approximately 43,000 patients annually, our 14-physician ED is affiliated with the Hospital Henri Mondor, a French university and tertiary-care 900-bed hospital located in the Paris area, serving a population of 800,000 individuals, and is equipped to perform percutaneous coronary reperfusion therapy.

Patients who came to the ED on weekdays (convenience sampling) between November 2002 and April 2004, with a suspicion of ACS, were eligible for enrollment. Symptoms prompting the suspicion of ACS included chest pain, dyspnea, epigastric pain, nausea–vomiting, neck or arm discomfort, fatigue, fall, syncope, and dizziness.[4,7,16] Symptoms refer to the main complaints that included persistent symptoms and symptoms that lasted 24 hours before ED arrival. Board-certified emergency physicians (EPs) attended all patients around the clock, and the attending physician made the symptoms-based decision to order cTnI testing.

Inclusion criteria for study patients were 1) age ≥ 18 years, 2) suspicion of ACS, and 3) order to measure cTnI. To meet Inclusion Criterion 2, a patient was required to present with one of the complaints listed above and either a cTnI level of ≥ 0.1 μg/L or at least two of the following: age 60 years or older, at least three cardiovascular risk factors, personal history of coronary artery disease, chest pain, or ECG changes indicating ischemia (ST-segment depression in two continuous leads, T-wave inversion > 3 mm, or transient ST-segment elevation). After giving informed consent for study participation, eligible patients were enrolled by attending EPs who then disclosed the allocated group.

Exclusion criteria were refusal or inability to provide informed consent or previous enrollment in the study. Because patients with ST elevation ACS (typical chest pain and persistent [> 20 min] ST-segment elevation [> 0.1 mV in limb leads or 0.2 mV in precordial leads]) did not represent a diagnostic challenge compared to NSTE-ACS and were candidates for urgent reperfusion procedure, they were excluded from the current analysis.[26] We defined two subgroups for analysis: ''high-risk patients'' (those with elevated cTnI) and ''low-suspicion'' ACS (neither chest pain nor a diagnostic ECG).

Study Protocol

Emergency physicians collected data through standardized patient interviews and medical record review during the ED evaluation. Data included 1) demographic data (i.e., age and gender); 2) clinical data (source of referral, presenting complaints, cardiovascular risk factors), personal and familial history of atherosclerosis, hypertension, hyperlipemia, diabetes mellitus, smoking history, body mass index ≥ 25 kg/m^2; 3) ECG abnormalities, thrombolysis in myocardial infarction (TIMI) score, and laboratory creatinine and cTnI; and 4) disposition (i.e., hospitalization, site of care).[4,13] Demographic data, presenting complaints, and cardiovascular risk factors were collected directly from patients' or a relative's interview. The source of the referral, ECG abnormalities, laboratory measures, and disposition were ascertained from the medical record.

Therapeutic data collected included whether anti-ischemic treatment (AIT) was ordered before cTnI results were available and, if so, whether the AIT was modified as a result of the cTnI. AIT refers to medication given in the ED to decrease acute myocardial ischemia, including antithrombin therapy (unfractionated heparin [UH] or low-molecular-weight heparin [LMWH]), platelet activation inhibitors (e.g., aspirin,

ticlopidine, clopidogrel), nitrates, and beta-blocking agents.

The study procedures occurred as follows: 1) cTnI measurement order; 2) enrollment and baseline data collection; 3) collection of blood sample; 4) POCT or CHLT of cTnI according to randomization; 5) request by physician for cTnI test result and, if necessary, order of a second cTnI measurement; 6) determination of therapeutic regimen and disposition; and 7) follow-up of procedures and medical outcomes. Each physician in charge of a patient was allowed to start AIT whenever necessary. This could happen before cTnI was ordered in the case of a perceived high-risk patient with NSTE-ACS or after becoming aware of the patient's cTnI.

On a daily basis, two independent physicians (PC, PM) not involved in patients' care, and two EPs in charge of study monitoring (AN, BR), reviewed the data collected for overall consistency, with a particular focus on the time of data collection. Discrepancies between patients' computer-based medical records that automatically write in memory the times of patient care at the ED (including arrival, first medical contact, cTnI availability, and treatment decision and disposition) were resolved by discussion with attending physicians and nurses. To assess whether treating physicians altered the randomization allocation, we compared the rank of subsequent numbered envelopes disclosure to the rank of patients' ED arrival time.

Measures

The primary study outcome measure was TAIT, as defined by time from presentation to time when any of the predefined AITs were ordered. Secondary study outcomes were the time from presentation to physician notification of cTnI level and the time from presentation to inpatient bed assignment (i.e., ED length of stay [LOS]).

For designated high-risk patients (NSTE-ACS and cTnI level ≥ 0.1 μg/L), performance of coronary angiography and reperfusion and 30- and 90-day mortality were assessed by two research assistants who were blinded to patients' allocation group, by standardized review of medical records and, whenever necessary, telephone interviews with the patient, a relative, or the family practitioner.

cTnI Level Measurement. Each patient's cTnI was measured after randomization allocation. Our hospital

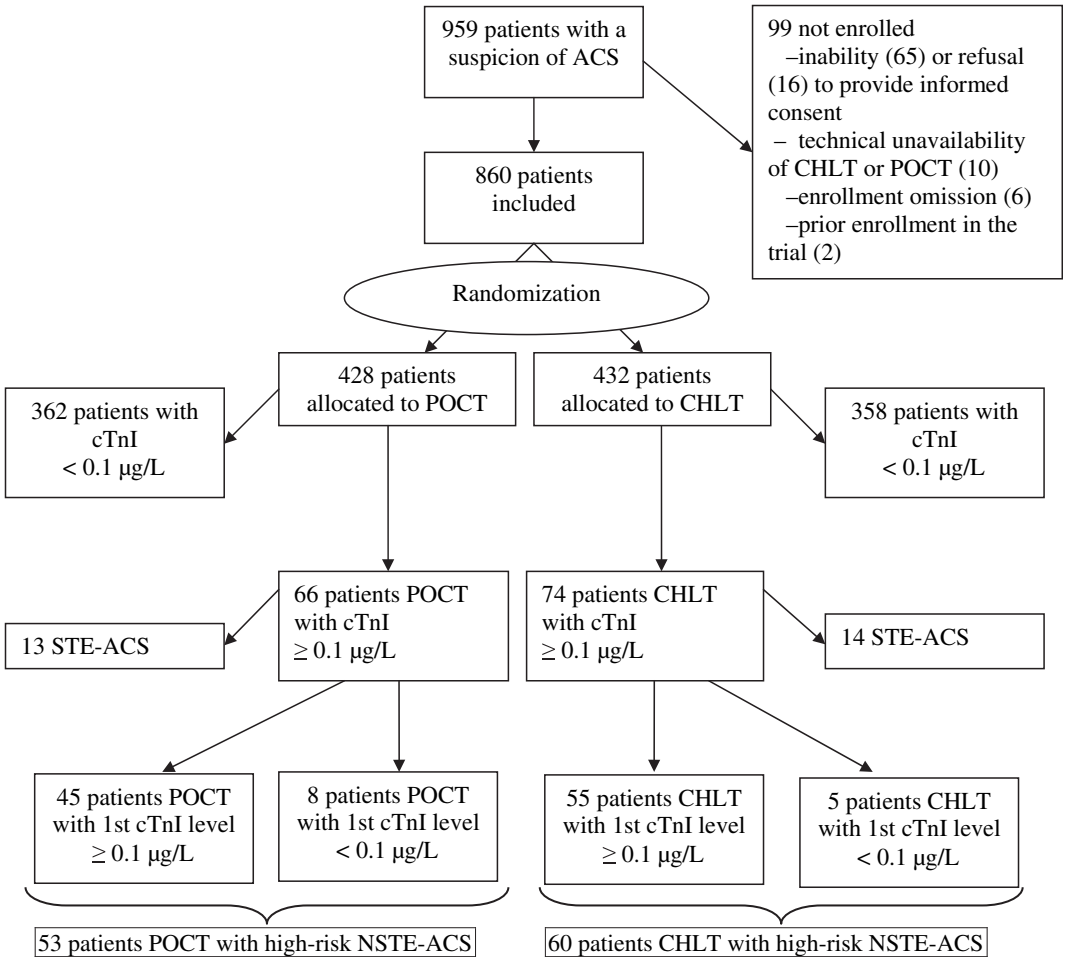

Figure 1. Study participants flow. ACS = acute coronary syndrome; CHLT = central hospital laboratory testing; cTnI = troponin I; NSTE-ACS = non–ST-segment elevation ACS; POCT = point-of-care testing; STE-ACS = ST-segment elevation ACS.

central laboratory staff took responsibility for cTnI measurement and for CHLT and POCT quality control per French quality standards for medical laboratories.[27] Based on the analytic performance of the Stratus CS system (Stratus CS test systems, Dade Behring, Marburg, Germany) and on consensus statements by the joint European Society of Cardiology/American College of Cardiology (ESC/ACC), all patients with cTnI ≥ 0.10 μg/L were considered positive.[28,29]

Point-of-care Testing. We used a whole-blood rapid-assay device (Stratus CS), which is a fluorometric enzyme immunoassay analyzer for quantitative determination of cTnI. The device analyzes closed routine sample tubes containing anticoagulated whole blood and gives results within 15 minutes.[29] The Stratus CS cTnI assay is appropriate for clinical use as it is diagnostically equivalent to CHLT (good correlation to CHLT and precision of a highly sensitive troponin

Table 1
Baseline Patients Characteristics

	Overall		Patients with cTnI ≥0.1 μg/L	
Characteristics*	POCT (*n* = 419)	CHLT (*n* = 414)	POCT (*n* = 53)	CHLT (*n* = 60)
Demographic characteristics				
Median age (IQR)	62 (49–75)	64 (50–77)	67 (63–71)	68 (64–73)
Male gender (%)	62.0	58.4	71.7	60.0
Source of referral (%)				
Out-of-hospital emergency care services	11.1	10.1	13.2	17.0
Ambulance	17.1	22.6	20.7	23.7
Personal	56.8	51.6	50.9	45.8
Other	14.9	15.7	15.1	13.6
Cardiovascular risk factors (%)				
Body mass index ≥25 kg/m^2	58.7	58.6	50.0	51.8
Hypertension	48.8	49.1	62.3	60.0
Diabetes mellitus	20.7	21.4	22.6	30.0
Hyperlipidemia	32.6	35.8	43.4	31.7
Smoking history	52.2	48.0	54.7	50.0
Familial history of atherosclerosis	25.1	27.1	28.3	21.7
Personal history of atherosclerosis	46.7	48.1	37.7	35.0
Presenting complaint (%)				
Chest pain	57.7	55.6	54.7	66.7
Typical chest pain	25.1	25.3	43.4	46.7
Typical left arm pain	13.2	16.4	26.4	25.0
General malaise	18.9	23.8	7.5	25.0
Dyspnea	32.3	32.6	47.2	35.0
Epigastric pain	8.1	8.4	3.8	15.0
ECG features (%)				
ST-segment depression or transient elevation	10.0	14.1	11.3	21.7
T-wave inversion	24.5	30.8	34.0	50.0
Pathologic Q-wave	15.1	10.4	13.2	11.7
Left bundle branch block	13.8	16.2	5.7	8.5
Arrhythmia	10.0	11.2	1.9	8.3
Biologic measures				
Renal failure* (%)	4.7	5.8	11.8	17.5
cTnI measure (μg/L), Median (IQR)	0.02 (0.01–0.04)	0.01 (0–0.05)	0.24 (0.14–1.00)	0.44 (0.18–2.10)
Second cTnI measure† (μg/L), Median (IQR)	0.02 (0.01–0.05)	0.01 (0–0.04)	0.13 (0.11–0.16)	0.14 (0.14–0.29)
Thrombolysis in myocardial infarction score (%)				
1	18.9	17.6	9.4	10.0
2	28.3	30.7	32.1	41.7
3	31.1	27.2	28.3	16.7
4	16.7	16.4	22.6	15.0
5–6	4.9	8.2	7.5	16.7

CHLT = central hospital laboratory testing; cTnI = troponin I; ECG = electrocardiogram; IQR = interquartile range; POCT = point-of-care testing.
*Renal failure denoted patients with creatinine level ≥120 μmol/L or creatinine clearance estimated by the Crockroft formula <30 mL/min when available.
†195 patients underwent a second cTnI measurement.

Table 2
Comparisons of Time Lags in Minutes (Median and Interquartile Range [IQR]) between Patients Allocated to the Point-of-care Testing (POCT) or to the Central Hospital Laboratory Testing (CHLT) for Cardiac Troponin

	Overall		
Characteristics	POCT (n = 419)	CHLT n = 414)	p-Value
Time (minutes), median (IQR)			
From presentation to blood sample collection	75 (70–80)	65 (60–70)	0.005
From blood collection to physician notification of first cTnI	38 (35–42)	109 (104–115)	<0.001
From Presentation to AIT	151 (139–162)	198 (187–210)	<0.001
Length of stay at ED (min), median (IQR)	309 (204–411)	307 (229–401)	0.99

Low-suspicion ACS referred to patients presenting no chest pain and non–ST-deviation NSTE-ACS with elevated cTnI.
cTnI = troponin I; ED = emergency department; IQR = interquartile range; NSTE-ACS = non–ST-segment elevation acute coronary syndrome; AIT = anti-ischemic treatment.

method [a coefficient of variation of < 10% at the 99th percentile of the reference population]).[30,31]

ED nurses were trained by Dade Behring staff prior to the study enrollment period and performed all point of care cTnI measurements. In an attempt to defeat a potential Hawthorne effect, nurses were asked not to deliver the cTnI result until a request was obtained from the attending physician.

Central Hospital Laboratory Testing. CHLT of cTnI was performed on the Dimension RxL-HM analyzer (Dade Behring), a one-step enzyme immunoassay based on two cTnI-specific monoclonal antibodies that gives a result in about 17 minutes.[32]

Data Analysis

The sample size estimate was chosen to detect a difference of 15 minutes in TAIT between the study subgroups of patients with cTnI ≥ 0.1 μg/L, which were expected to include 15% of the entire study cohort. We assumed a power of 80%, statistical significance level of 5%, and mean TAIT of 90 minutes with standard deviation of 30 minutes, resulting in an estimated sample size of 64 patients in each high-risk subgroup that required 420 patients to be included in each study group.

Comparisons between groups were performed using the t-tests or Wilcoxon rank-test for continuous variables and chi-square or Fisher's exact test for categorical variables. All analyses were performed using Stata Version 8.0 (Stata Corp., College Station, TX).

RESULTS

Of the 959 patients screened from November 2002 to April 2004, a total of 833 were enrolled, of whom 419 (50.3%) were randomly allocated to the POCT group and 414 (49.7%) to the CHLT group (Figure 1). There were 53 (12.6%) high-risk patients in the POCT group and 60 (14.5%) in the CHLT group. Among those high-risk patients, 55 patients had a low suspicion of ACS, 27 (6.4%) in the POCT group and 28 (6.8%) in the CHLT group.

Troponin I was elevated in 113 (13.1%) patients (53 [46.9%] in the POCT group and 60 [53.1%] in the CHLT group; Table 1). Ninety-nine (87.6%) patients were classified positive on the first cTnI test, and 14 (12.4%) more were positive on the second test. Of those, 85 (75.2%) patients were treated with platelet activation inhibitors (aspirin alone in 70.8%), 40 (35.4%) with UH, 28 (24.8%) with LMWH, 40 (35.4%) with nitrates, and 4 (3.5%) with beta-blocking agents at ED. Treatment was started in 54 (47.8%) patients before physician knowledge of cTnI: 35 (64.8%) of these patients reported chest pain and 30 (55.6%) had ECG abnormalities suggestive of acute ischemia. AIT given before cTnI knowledge included aspirin, 33 of 54 (61.1%); nitrates, 25 of 54 (46.3%); UH, 20 of 54 (37.0%); and LMWH, 12 of 54 (22.2%).

The median time from patient arrival in the ED to blood sample collection was 10 minutes longer in the POCT group than in the CHLT group for the 833 patients with a suspicion of NSTE-ACS, but this difference in times was smaller in the study subgroups (Table 2). The median time from blood sample collection to physician notification of cTnI was shorter in the POCT group than in the CHLT group. As a result, TAIT was shorter for POCT patients than for CHLT patients, and this was even more pronounced for the two study subgroups. Excluding those high-risk patients who only had an elevated troponin on the second sample (first troponin normal), the results were similar (TAIT, 102, interquartile range [IQR] = 84–148 vs. 166, IQR = 123–216) for POCT and CHLT, respectively [p < 0.001]. Median LOS in the ED did not significantly differ in any of the three POCT-CHLT comparisons.

Patients with elevated cTnI level usually became inpatients (94.7%); 78 (69.0%) were initially admitted to a cardiac or medical intensive care unit (Table 3), and 3 patients refused hospitalization against medical advice. Procedures did not differ by study group: 52 (46.0%) patients had a coronary angiography during the index hospitalization, of whom 5 (4.4%) had immediate coronary angiography (ICA), 20 (17.7%) had transluminal coronary angioplasty, and 15 (13.3%) had a coronary artery bypass graft. The 5 patients with ICA reported typical chest pain on arrival. Two (1.8%) patients, both from the POCT group, died within 30 days after ED arrival. Their initial cTnI levels were 0.1 and 0.42 μg/L.

NSTE-ACS Elevated cTnI			Low-suspicion ACS		
POCT (n = 53)	CHLT (n = 60)	p-Value	POCT (n = 27)	CHLT (n = 28)	p-Value
70 (54–87)	67 (52–82)	0.77	57 (39–110)	61 (27–112)	0.98
36 (28–43)	101 (90–113)	<0.01	27 (15–60)	120 (76–137)	<0.01
145 (117–173)	192 (163–221)	<0.01	120 (92–248)	198 (156–244)	0.03
275 (161–422)	199 (122–353)	0.15	226 (163–469)	201 (115–428)	0.43

Table 3
Treatment and Medical Outcomes of Patients with NSTE-ACS and cTnI ≥ 0.1 μg/L

	POCT (n = 53)	CHLT (n = 60)	p-Value
Anti-ischemic treatment at the ED (%)			
Overall	84.9	85.0	0.99
Specific treatment prior to physician notification	41.5	53.3	0.21
Antiplatelet therapy (%)			
Overall	77.4	73.3	0.62
Prior to physician notification	24.5	33.3	0.30
Unfractionated heparin (%)			
Overall	30.2	40.0	0.23
Prior to physician notification	13.2	21.7	0.24
Low-molecular-weight heparin (%)			
Overall	30.2	20.0	0.21
Prior to physician notification	11.3	10.0	0.82
Nitrates (%)			
Overall	43.4	28.3	0.09
Prior to physician notification	18.9	25.0	0.43
Beta-blocking agents (%)			
Overall	5.7	1.7	0.34
Prior to physician notification	0	0	–
Procedures (%)			
Coronary angiography	47.2	45.0	0.64
Immediate coronary angiography	1.9	6.7	0.22
Transluminal coronary angioplasty	17.0	18.3	0.42
Aortocoronary bypass	11.3	15.0	0.57
Outcome (%)			
Inpatient treatment	96.2	93.3	0.68
Hospitalization in cardiology wards	69.8	68.3	0.86
Admission in CCU or ICU	67.9	70.0	0.81
30-day mortality*	3.8	0.0	0.21
90-day mortality*	11.5	8.3	0.57

CCU = coronary care unit; CHLT = central hospital laboratory testing; cTnI = troponin I; ICU = intensive care unit; NSTE-ACS = non–ST-segment elevation acute coronary syndrome; POCT = point-of-care testing.
*Outcomes were missing for mortality at 30 days (n = 1) and mortality at 90 days (n = 1) in the CHLT group.

By the 90-day follow-up, overall 11 (9.8%) patients had died; their median cTnI level was 1.1 μg/L (IQR = 0.24–2.51 μg/L) vs. 0.29 μg/L (IQR = 0.17–1.2 μg/L) in survivors (p = 0.34). Neither 30-day mortality nor 90-day patient mortality rates differed between study groups.

DISCUSSION

In this trial of adults arriving at the ED with suspicion of NSTE-ACS, we found that POCT was associated with a faster decision-making process than CHLT was. POCT was associated with a shorter TAIT (median 151 min, IQR = 139–162 min) compared to CHLT (median 198 min, IQR 187–210 min). Therefore, the diagnosis of myocardial infarction could be made slightly earlier in the subset of patients with vague symptoms (Figure 2).

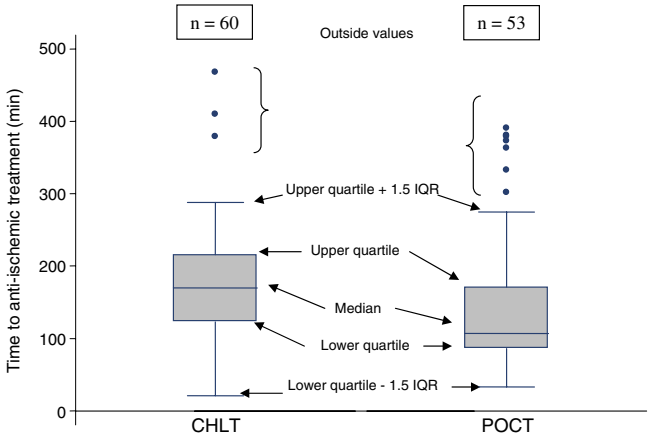

Figure 2. Time to anti-ischemic therapy (TAIT) of the 113 patients with non–ST-segment elevation acute coronary syndrome and elevated troponin I (cTnI; ≥ 0.1 μg/L) allocated to point-of-care testing (POCT) compared to those allocated to central hospital laboratory testing (CHLT) for patients with NSTE-ACS and elevated cTnI. Boxes are delimited by the upper limit (75th percentile) and by the lower limit (25th percentile) of the interquartile range (IQR). The line inside boxes figures the median value of the TAIT. Adjacent lines figure the upper (upper quartile + 1.5 IQR) and the lower (lower quartile – 1.5 IQR) adjacent values, and dots represent outside values.

As there is increasing evidence supporting early invasive therapy for patients with high-risk NSTE-ACS, we believe that a time saving of 45 minutes has clinical relevance in the ED.[2,3,7,33] Nonetheless, our findings underscore the limited impact of POCT for shortening ED LOS.

Consistent with other reports, numerous study patients (45.1%) did not have typical chest pain.[7,34] Presenting complaints differed somewhat, particularly within the study subgroups. Consistent with current European guidelines, AIT was often started in the ED in both groups.[4,10,35] However, AIT was initiated in 52.2% of high-risk patients after cTnI level knowledge, and this rate was somewhat higher in the POCT group. Accordingly, ICA was less likely performed on POCT patients. The reliability of cTnI levels obtained on the POCT device has been repeatedly demonstrated.[23,24,36] Therefore, the differences in early onset of AIT and in ICA performance between study groups likely reflects the higher rate of POCT patients arriving with vague symptoms and nonsuggestive ECG findings, as has been associated with the underuse of invasive procedures.[7,35] Most high-risk patients were admitted to coronary care or intensive care units and 46.0% subsequently underwent coronary angiography followed by revascularization procedure. This underscores the coronary artery disease severity and the risk of adverse outcomes associated with delayed treatment of high-risk patients, who are often undertreated and are at greater risk of death.[7,33,37,38]

Point-of-care testing resulted in shortening the TAIT, particularly for 38.9% of high-risk patients with a low suspicion of ACS. For nontroponin testing, previous studies have not always shown a significant benefit of implementing POCT in the ED.[21,39–42] By studying only patients with suspicion of NSTE-ACS, for whom treatment decision or bed request may be delayed until the cTnI result is known, we were able to show a difference in TAIT.[23,43]

Nevertheless, despite hastening decision-making, we did not demonstrate a significant difference in the study group average LOS in the ED. This suggests that POCT is only part of the whole-system approach that is required to improve timeliness of care.[21–23] Indeed, many other factors determine the duration of ED visits, such as the absence of centralized bed assignment in our hospital. Apart from chance variation, we could not explain the unexpected trend toward a longer LOS for the POCT group. Nonetheless, even if EPs cannot entirely control delays from the onset of symptoms to hospital admission, reducing in-hospital care delays can reduce both morbidity and mortality. Depending on the institution, a shorter time to diagnosis might also affect time to bed request or unit assignment.[22,44]

LIMITATIONS

We were not able to enroll patients throughout the entire week, which may have introduced a selection bias. Physicians and nurses could not be blinded to patient assignment, which may have influenced their attitudes or recording of their decision-making times. To decrease the chance of this bias, data collection was reviewed by nonstudy physicians for overall consistency with a particular focus on the time of data collection. Our study was not powered to detect differences in mortality, particularly regarding high-risk patients, although time saving itself has proven to be a relevant endpoint.[33,44,45] The quite low and late AIT use, particularly for high-risk patients, may have artificially increased the proportion of patients for whom the cTnI level was the time-limiting step in the diagnosis of low suspicion ACS.[22,46] Because timeliness of care is tightly linked to routine practices, and the impact of POCT may vary across institutions, and because our findings were obtained from a single center trial, our results might not be generalizable to other EDs. Finally, inasmuch as the crude cost of POCT for cTnI was higher than CHLT, and the fact that we did not collect the working time spent by nurses to perform the procedure, additional studies should address the cost-effectiveness of POCT for cTnI measurement in the ED.[47,48]

CONCLUSIONS

Point-of-care testing decreased the time to the initial delivery of AIT for ED patients with suspicion of NSTE-ACS, both overall and in the high-risk group. This benefit may be particularly relevant for NSTE-ACS patients with low suspicion, for which time to diagnosis is shortened. This decreased time to delivery of therapy did not significantly impact ED LOS and outcomes in this small study.

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