Effectiveness of Point-of-Care High-Sensitivity Troponin Testing in the Emergency Department: A Randomized Controlled Trial

Viola I. L. Thulin, MD; Silje M. F. Jordalen, MD; Gard M. S. Myrmel, MD; Ole Christian Lekven, MD; Jeyaseelan Krishnapillai, MD; Ole Thomas Steiro, MD, PhD; Richard Body, MD, PhD; Paul Collinson, MD; Fred S. Apple, PhD; Louise Cullen, MD, PhD; Tone M. Norekvål, PhD; Torbjørn Wisløff, MSc, PhD; Kjell Vikenes, MD, PhD; Rune O. Bjørneklett, MD, PhD; Torbjørn Omland, MD, PhD; Kristin M. Aakre, MD, PhD*

*Corresponding Author. E-mail: Kristin.moberg.aakre@helse-bergen.no.

Study objective: To compare the effectiveness of high-sensitivity cardiac troponin (hs-cTn) point-of-care testing to central laboratory hs-cTn measurements when investigating patients presenting to the emergency department (ED) with symptoms of acute coronary syndrome.

Methods: The WESTCOR point-of-care study was a single-center prospective randomized controlled trial where we randomized patients presenting with possible acute coronary syndrome in a 1:1 fashion to receive either 0/1-hour centralized hs-cTnT measurements (control) or 0/1-hour point-of-care hs-cTnI testing (intervention). We defined length of stay (LOS) in the ED as the primary endpoint and the minimum clinically meaningful difference as 15 minutes.

Results: We included 1,494 patients in the final analysis, 728 in the point-of-care group, and 766 in the control group. The median (interquartile range) age was 61 (22) years, and 635 (42.5%) were women. Median LOS in the ED was 174 (95% confidence interval [CI] 167 to 181) and 180 (95% CI 175 to 189) minutes in the point-of-care and control group, respectively, resulting in a reduction in median LOS of 6 minutes (95% CI −4 to 17). Acute myocardial infarction, death, or acute revascularization occurred in 83/728 (11.4%) of point-of-care and 72/766 (9.4%) of control patients.

Conclusions: We found that implementing point-of-care hs-cTnI testing in the ED with a 0/1-hour diagnostic algorithm did not lead to a clinically meaningful reduction in ED LOS. We observed no difference in the incidence of myocardial infarction, acute coronary revascularization, or death during 30 days follow-up. [Ann Emerg Med. 2025;86:124-135.]

Please see page 125 for the Editor's Capsule Summary of this article.

Keywords: Accelerated diagnostic protocol, Chest pain, Cardiac biomarkers, Acute coronary syndrome, Emergency department crowding.

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INTRODUCTION

Background

Emergency department (ED) crowding is a global problem, associated with increased cost, morbidity, and death.[1-3] Symptoms suggestive of acute coronary syndrome represent one of the most common reasons for presentation to the ED.[4-6] Only 5% to 20% of patients presenting with these symptoms are finally diagnosed with acute coronary syndrome, the majority have benign conditions not requiring hospital admission.[7-15] The development of high-sensitivity cardiac troponin (hs-cTn) assays has paved the way for implementation of accelerated diagnostic protocols. These protocols include troponin measurements at admission and after 1 to 2 hours, allowing for early risk prediction.[7-13] Accelerated diagnostic protocols have been proven safe and efficient, being able to significantly reduce length of stay (LOS) in the ED and are currently recommended by the European clinical practice guidelines and supported by the American guidelines.[16-29]

Importance

Until recently, hs-cTn assays have only been available on central laboratory platforms. Studies rarely report data on total turnaround time for these assays, and although newer short turnaround time (STAT) assays have an analytical turnaround time as low as 9 minutes' laboratories may

Editor's Capsule Summary

What is already known on this topic

High-sensitivity troponin measurements can quickly detect acute myocardial infarction.

What question this study addressed

Does a 0/1-hour protocol using point-of-care troponin versus 0/1-hour laboratory testing reduce emergency department (ED) length of stay?

What this study adds to our knowledge:

The point-of-care troponin did not lead to a clinically meaningful reduction in ED length of stay or any difference in 30-day events.

How this is relevant to clinical practice:

Quick turnaround times related to point-of-care testing may not translate into reduced length of stay.

struggle to reach the recommended less than 60 minutes target for total troponin turnaround time due to pre- and post-analytical delays.[30] Point-of-care hs-cTn assays providing shorter total turnaround times are now available.[31] The analytical quality and clinical performance of these assays have been documented, and recent papers suggest single sample, 0/1-hour, and 0/2-hour point-of-care accelerated diagnostic protocols may be used safely.[32-38] However, it is currently unknown if hs-cTn testing by point-of-care instruments can be used to improve patient care in the ED.

Goals of This Investigation

We designed the WESTCOR point-of-care study to determine whether incorporating hs-cTnI measurements using a point-of-care instrument (Atellica VTLi, Siemens Healthineers) in a 0/1-hour accelerated diagnostic protocol could reduce LOS in the ED compared with a 0/1-hour accelerated diagnostic protocol using central laboratory hs-cTnT testing. We hypothesized that the point-of-care algorithm would reduce ED LOS compared with currently recommended accelerated diagnostic protocols using central laboratory testing.

MATERIALS AND METHODS

Study Design and Setting

We conducted this single-center prospective randomized clinical trial at Haukeland University Hospital in Bergen, Norway. The ED staff included a mix of emergency physicians, emergency medicine trainees, and junior doctors from other specialties. The ED only receives adult (>18 years) medical patients. In 2022, approximately 3,000 patients presented with symptoms suggestive of acute coronary syndrome, accounting for 6.7% of all ED visits. ED LOS for patients with chest pain at our ED is approximately 3 hours. A central laboratory hs-cTnT assay is used in standard practice at our hospital, and approximately 71% to 91% of results are reported within 60 minutes of arrival at the central laboratory. We did not perform any blinding of the groups after inclusion; hs-cTnT and point-of-care hs-cTnI were reported as per group.

This study followed the principles of the Declaration of Helsinki and has received approval from the regional ethics committee (REC number 285544); it is registered at ClinicalTrials.gov (NCT05354804) and is reported in accordance with the CONSORT statement. We have previously published the study protocol describing the study design.[39]

Study Enrollment

All patients 18 years of age and older presenting to the ED from March 14, 2022 to March 26, 2024 with symptoms suggestive of acute coronary syndrome were eligible for inclusion. Exclusion criteria were as follows: (1) STEMI (ECG criteria), (2) clinically unstable patients in need of immediate cardiac catheterization, (3) patients with significant comorbidities likely to compromise short-term survival (<2 months expected survival), (4) patients transferred from other hospitals; and (5) patients who were unable to provide informed consent. We applied a modified intention-to-treat principle, and patients who had previously been included in the study, those transferred to another hospital's ED, and those missing the allocated 0-hour troponin measurement were excluded from this analysis.

Study nurses without formal laboratory training performed consecutive enrollment after capacity, obtained the blood samples, and performed the point-of-care analyses in the ED. The study nurses obtained oral consent before inclusion and randomized the patients immediately after by simple parallel randomization in concealed envelopes in a 1:1 fashion. They collected written consent when the clinical situation was stabilized. Patients could withdraw from the study at any time. The Department of Research and Development at Haukeland University Hospital, Bergen, Norway, provided the random allocation sequence.

Control arm. The study nurses collected blood samples from patients, randomized to the control arm, on

presentation and 1 hour later. We measured hs-cTnT using the Roche Diagnostics STAT assay, with an analytical turnaround time of 9 minutes. The limit of detection for this assay is 3 ng/L, sex-specific 99th percentile of 9 ng/L (women) and 17 ng/L (men), and 10% within-series analytical coefficient of variation (CV_A) of 4.5 ng/L.[32] We used assay-specific 0/1-hour rule-out cutoffs from the European Society of Cardiology (ESC) guidelines (hs-cTnT less than 12 ng/L and 1-hour delta of less than 3 ng/L) to indicate low risk.[28] To guide the high risk of non-ST-segment elevation myocardial infarction (NSTEMI), we used the rule-in cutoffs (hs-cTnT more than 52 ng/L or 1-hour delta of 5 ng/L or more). Laboratory assays and cutoffs are also presented in Figure 1. The accelerated diagnostic protocols did not suggest a single-sample rule-out, which represented a pragmatic local adaption to the ESC algorithm, as we expected the total turnaround time for hs-cTnT to be 60 minutes or more. We recommended a 3-hour hs-cTnT sample in all patients with less than 1 hour from symptom debut, ongoing recurrent symptoms, and in patients without allocation to the "rule-out" or "rule-in" categories after the 0- and 1-hour sample as suggested by the ESC 0/1-hour algorithm.[28] All hs-cTnT and standard laboratory tests were measured in the central hospital laboratory. We suggested rapid discharge for patients who were "ruled out" by the hs-cTnT 0/1-hour algorithm, had a History, Electrocardiogram, Age, Risk factors, and Troponin score less than 4, and no suspicion of any serious noncoronary diagnosis. Patients were treated, admitted, or discharged based on the decision of the attending physician.

Intervention arm. Patients randomized to the intervention arm were treated similarly to those in the control arm, except that the presentation and 1-hour sample were measured in the ED using the Atellica VTli point-of-care hs-TnI instrument from Siemens Healthineers with an analytical turnaround time of 8 minutes. We reanalyzed the point-of-care samples up to 2 times if a measurement error was reported. The limit of detection for this assay is 1.6 ng/L, sex-specific 99th percentile of 18 ng/L (women) and 27 ng/L (men), and 10% CV_A of 8.9 ng/L.[32] We determined the low-risk cutoffs based on a publication from Apple et al,[36] suggesting a single-sample rule-out of 4 ng/L and additional reviews of the correlation with other central laboratory hs-cTnI (Siemens Atellica and Abbott Alinity) and hs-cTnT (Roche Diagnostics) assays for which 0/1 hour algorithms have been suggested.[39] As a higher cutoff is applicable when 2 samples are obtained, we settled on a baseline rule-out concentration of hs-cTnI less than 6 ng/L with a 1-hour delta less than 3 ng/L.[28] This baseline cutoff was later confirmed in a publication by Cullen et al.[35] No 0/1-hour rule-in algorithm had been published during the planning of the study, and accordingly, we took a conservative approach, and clinicians were informed that concentrations above the 99th percentile signaled an increased risk of NSTEMI. We also analyzed the admission hs-cTnT measurements in the central laboratory and made them

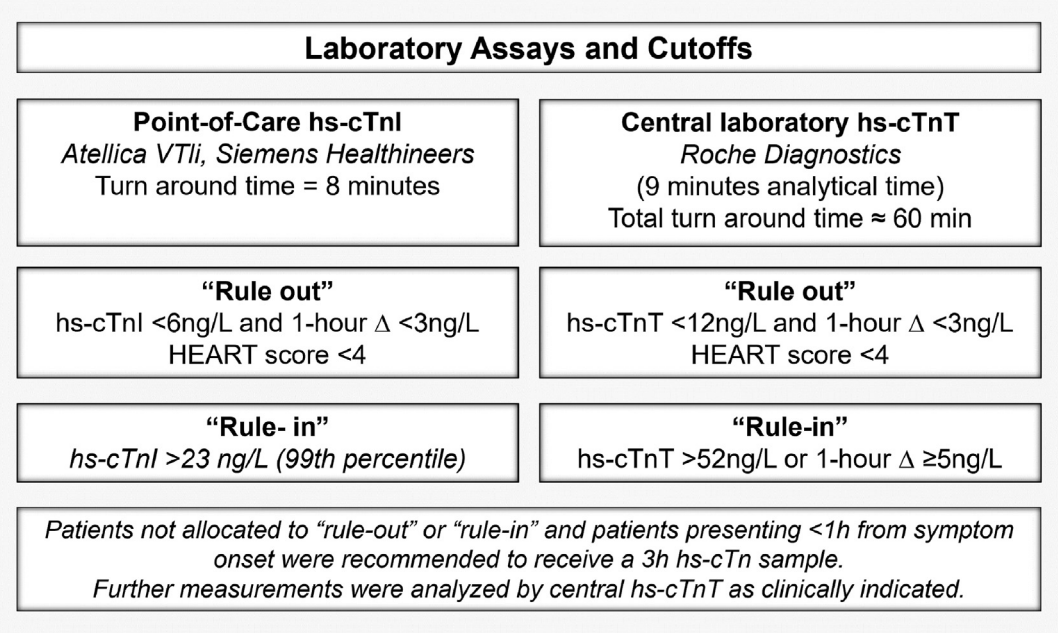

Figure 1. Illustration of laboratory assays, turnaround time, and diagnostic rules.

available on special request if they were needed for comparison.

Outcomes

We discussed different endpoints during the planning of the study and initially defined one primary endpoint for each type of research topic evaluated in this study, eg, ED effectiveness, safety, costs, and patient satisfaction. Based on feedback from some of the scientific advisers, this was changed into a single primary endpoint (LOS in the ED) and multiple secondary endpoints to be adherent to the CONSORT guideline.

The secondary effectiveness endpoints were percentages discharged within 3 hours, 6 hours, and total hospital LOS. The secondary safety endpoints were as follows: (1) all-cause mortality and acute myocardial infarction (AMI) (with and without index events) within 30 days and (2) all-cause mortality, AMI, and acute unplanned revascularization within 30 days (with and without index events). The rate of readmission by any cause within 30 days was prespecified as another outcome (not primary nor secondary). Data from the remaining prespecified secondary outcomes have not yet been analyzed or are currently unavailable. These are planned to be published in the following 2 future articles: (1) patient satisfaction measured by 3 validated questionaries and total episode cost; (2) total cost related to all hospital contacts during 12 months follow-up; and (3) death, myocardial infarction, and acute revascularization within 12 months from inclusion.

We collected baseline characteristics and 30-day safety data through review of patient case note files. These files provide clinical information from our health care trust and additional data on hospital admissions or death on a national level. LOS data were collected from the hospital logistics software that registers the actual time the patient arrives, is physically transferred, or physically leaves the department.

Two independent cardiologists adjudicated the index diagnosis based on all available clinical data, including laboratory results, ECG, echocardiography, and other imaging findings that were available up to 30 days after inclusion using prespecified diagnostic criteria. We used hs-cTnT for adjudication in all patients (control and intervention arms) as a laboratory analytical platform for hs-cTnI testing was not available in our central laboratory. Unstable angina pectoris and NSTEMI diagnoses were adjudicated based on the Fourth Universal Definition of Myocardial Infarction using sex-specified hs-cTnT cut offs of more than 9 ng/L and more than 17 ng/L and a delta value of more than 50% (minimum absolute deltas of >5 ng/L in women and >9 ng/L in men), if the baseline concentration were below the 99th percentile of the upper reference limit, and 20% if it was above.[40,41]

Statistical Analysis

We reported categorical variables as numbers and percentages and continuous variables as median with interquartile range (IQR) or 95% confidence intervals (CIs) as data were not normally distributed. The normality of distribution was assessed with Kolmogorov-Smirnov and Shapiro-Wilk tests for normality. We compared the between-group differences in the primary outcome (LOS in the ED) using the Mann-Whitney *U* test and evaluated the secondary composite safety outcomes (30-days AMI and death and 30-days AMI, death, and revascularization with and without index events) by estimating the odds ratio using logistic regression. No additional covariates were included in the logistic regression mode. We generated Kaplan-Meier survival plots and compared them using the log-rank test.

The database (WESTCOR-POC_version 1) was locked on June 12, 2024 and saved on a secure research server provided by the Haukeland University Hospital. The statistical analysis was performed using SPSS Statistics (version 26.0), MedCalc software Ltd (version 17.6), and R (version 4.1.2) by 2 independent researchers (IVLT and GMSM); GMSM was blinded toward group randomization.

Power Calculation

We estimated the control arm to have a LOS in the ED of 180 (SD 90) minutes. To have a power of 0.80 and alpha of 0.05 (independent sample *t* test), 190 and 566 patients needed to be included in each arm for a difference of 30 or 15 minutes, respectively. Based on information from the iCarefaster study made available to us during the planning of the study and later published, we expected the difference between the groups to be 15 to 30 minutes.[42]

RESULTS

Study Subjects

We enrolled 1,614 patients with symptoms suggestive of acute coronary syndrome during the 2-year period. Fourteen patients met the exclusion criteria, 798 were randomized to the point-of-care group, and 802 to the control group, illustrated in Figure 2. Fourteen patients withdrew from the study after inclusion. We excluded 92 patients from the final analysis due to prior inclusion (point-of-care n=9 and control n=6), transferal to another ED (point-of-care n=4, control=7), or incomplete 0 h

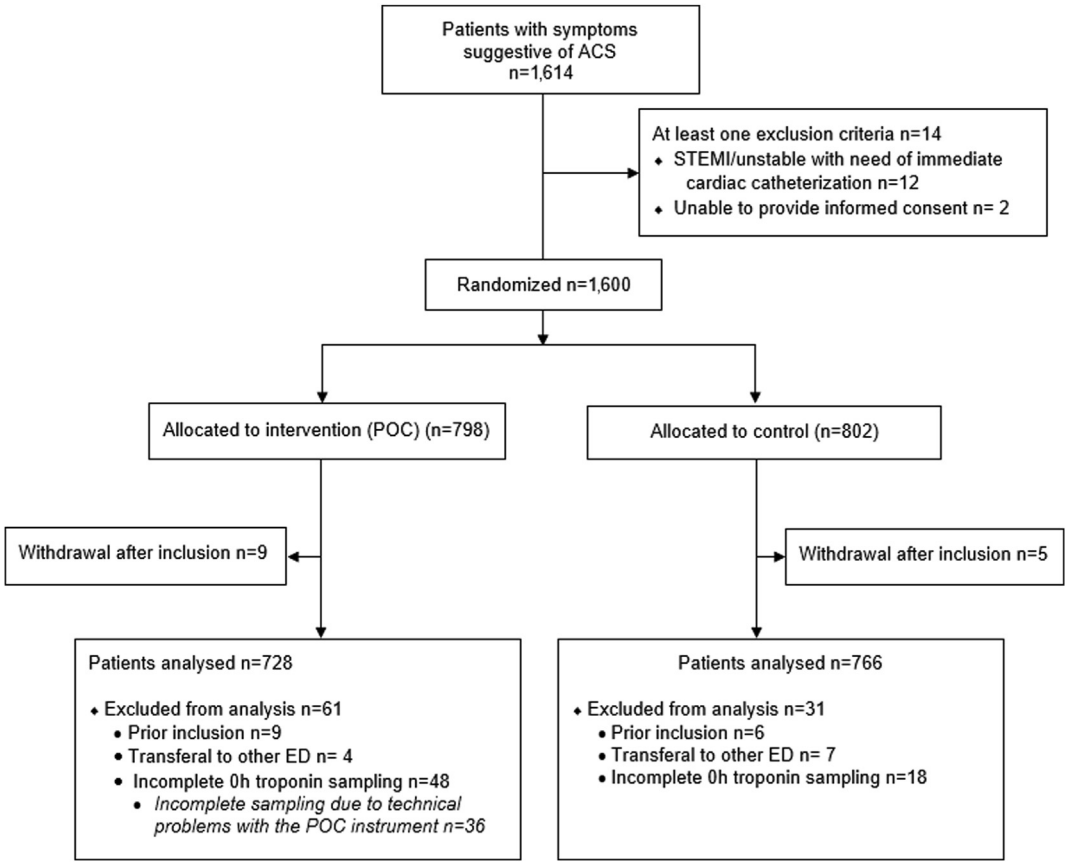

Figure 2. Outline of patient inclusion, exclusion, and total study group. *POC*, Point-of-Care; *STEMI*, ST-elevation myocardial infarction.

troponin sampling (point-of-care n=48, control n=18), leaving 1,494 patients in the final analysis (point-of-care n=728, control n=766). Thirty-six of the 48 patients in the point-of-care group were excluded from the analysis due to incomplete 0 h troponin sampling, which was caused by technical problems with the point-of-care instrument. No patients were lost to follow-up.

The included patients had a median age of 61 years (IQR, 22 years), and 635 (42.5%) were women. Among them, 238 (16%) were early presenters (<3 hours from symptom onset), and 20 (1.3%) were very early presenters (<1 hour from symptom onset). Index NSTEMI was observed in 79 out of 1,494 patients (5.3%). Patient baseline characteristics and adjudicated diagnoses during index hospitalization were similar between groups (Table 1).

Main Results

We observed a median ED LOS of 174 (95% CI 167 to 181) and 180 (95% CI 175 to 189) minutes in the point-of-care and control group, respectively (*P*=.024) (Table 2), resulting in a reduction in median LOS of 6 minutes (95% CI −4 to 17). Similar proportion of patients were discharged within 3 hours and 6 hours in both groups: 16% (n=115/728, 95% CI 13 to 19) (point-of-care) versus 15% (n=113/766, 95% CI 12 to 17) (control) and 38% (n=273/728, 95% CI 34 to 41) (point-of-care) versus 38% (n=290/766, 95% CI 35 to 41). Total hospital LOS also remained similar between groups, with a median of 21 hours (95% CI 19 to 22) (point-of-care) versus 20 hours (95% CI 18 to 21) (control).

We registered death or AMI within 30 days in 51 (7.0%, 95% CI 5.2 to 8.8) patients in the point-of-care group and 39 (5.1%, 95% CI 3.5 to 6.7) patients in the control group (Table 3 and Figure 3). Sixty-one (8.4%, 95% CI 6.0 to 10.0) patients in the point-of-care group and 63 (8.2%, 95% CI 6.0 to 10.0) patients in the control group underwent acute revascularization. After excluding index events, we observed a composite outcome of death or AMI in 5 out of 728 patients (0.7%, 95% CI 0.1 to 1.3) (point-of-care) versus 2 out of 766 patients (0.3 %, 95% CI −0.1 to 0.6) (control). We observed acute revascularization in 2 out of 728 patients (0.3%, 95% CI 0.0 to 1.0) (point-of-care) versus 3 out of 766 patients (0.4%, 95% CI 0.0 to 1.0) (control). Sixty out of 728

Table 1. Baseline characteristics and adjudicated index diagnosis.

Characteristic	POC (n=728)	Control (n=766)
Age (y) median (IQR)	61 (23)	61 (22)
Age (y) ≥80 (%)	75 (10.3)	83 (10.8)
Sex, female (%)	310 (42.6)	325 (42.4)
Source of referral, n (%)		
Direct	7 (1.0)	14 (1.8)
Ambulance	98 (13.5)	94 (12.3)
Out-of-hospital emergency care clinic	300 (41.2)	293 (38.3)
General practitioner	277 (38.0)	311 (40.6)
Time to presentation, n (%)		
<1 h	7 (1.0)	13 (1.7)
< 3 h	117 (16.1)	121 (15.8)
≥ 3 h	610 (83.9)	643 (84.2)
Presenting symptom, n (%)		
Chest pain	405 (55.6)	438 (57.2)
Chest tightness/pressure	262 (36.0)	248 (32.4)
Dyspnea	20 (2.7)	31 (4.0)
Others	41 (5.6)	49 (6.4)
Clinical findings		
Systolic blood pressure (mmHg), median (IQR)	145 (28)	144 (27)
Pulse (beats/min), median, (IQR)	72 (19)	72 (20)
eGFR (mL/min/1.73m^2), median, (IQR)	92 (22)	91 (26)
- eGFR<60 mL/min/1.73m^2 (%)	55 (7,6)	70 (9,1)
Total cholesterol (mmol/L), median (IQR)	4.8 (1.8)	4.7 (2.0)
LDL cholesterol mmol/L), median (IQR)	3.0 (1.7)	2.9 (1.8)
BMI, median (IQR)	27 (6)	27 (6)
ECG-significant ST deviation, n (%)	12 (1.6)	18 (2.3)
ECG–nonspecific repolarization disturbance, n (%)	88 (12.1)	78 (10.2)
HEART score, median (IQR)	3 (3)	4 (3)
- HEART score >4, n (%)	379 (52.1)	379 (49.5)
Suspicion of serious noncoronary condition, n (%)	147 (20.2)	164 (21.4)
Medical history n (%)		
Hypertension	322 (44.6)	349 (45.6)
Hyperlipidaemia*	341 (46.8)	368 (48.0)
Diabetes mellitus	85 (11.7)	99 (12.9)
Family History of atherosclerosis†	203 (39.1)	206 (39.0)
Current smoker	106 (14.8)	107 (14.3)
Personal history of atherosclerosis	204 (28.0)	205 (26.8)
Previous AMI	127 (17.4)	122 (15.9)
Previous PCI/CABG	152 (20.9)	163 (21.3)
Previous stroke	32 (4.4)	28 (3.7)
Peripheral artery disease	17 (2.3)	16 (2.1)
ED patient flow metrics		
Minutes from presentation to first troponin sample, median (IQR)	13 (9)	12 (10)
Minutes from presentation to first reported troponin result, median (IQR)	36 (16)	68 (17)
Minutes from presentation to reported 1 h troponin result, median (IQR)	95 (19)	128 (21)
Minutes from presentation to evaluation by physician‡, median (IQR)	34 (64)	36 (62)
Proportion with 1h sample within 60 +/-10 min, n (%)	635 (87.2)	667 (87.1)
Proportion of patients randomized to POC with central laboratory hs-cTnT reported in the ED, n (%)	203 (29.9)	NA
Proportion of patients randomized to POC discharged from the ED with central laboratory hs-cTnT reported in the ED, n (%)	65 (21.9)	NA
Management, n (%)		
Discharge from ED	297 (40.8)	305 (39.8)
Chest radiography in the ED	269 (37.0)	324 (42.3)
Echocardiogram in the ED	325 (44.6)	346 (45.2)
CT Coronary Angiography Index admission	204 (28.0)	222 (29.0)
CT coronary angiography outpatient referral	41 (5.6)	46 (6.0)
Coronary angiography during index admission	111 (15.2)	103 (13.4)
Risk group allocation by hs-cTn ADP§, n (%)		
"Rule-out"	277 (38.5)	489 (64.1)
"Rule-in"	75 (10.4)	60 (7.9)
No "rule-out" or "rule-in" allocation	367 (51.0)	214 (28.0)
Adjudicated index diagnosis, n (%)		
NSTEMI	44 (6.0)	35 (4.6)
Type 1 AMI	40 (5.5)	33 (4.3)
Type 2 AMI	4 (0.5)	1 (0.3)
Unstable angina pectoris	45 (6.2)	43 (5.6)
Stable angina pectoris	32 (4.4)	33 (4.3)
Other cardiac disease	36 (4.9)	43 (5.6)
Other noncardiac disease	99 (13.6)	112 (14.6)
Unspecified chest pain	472 (64.8)	500 (65.3)
Troponin concentrations, median (IQR)		
hs-cTnI at admission	6.7 (6.3)	NA
hs-cTnT at admission	NA	8.0 (10.0)
hs-cTnI at one hour	6.9 (6.1)	NA
hs-cTnT at one hour	NA	8.0 (10.0)

ADP, accelerated diagnostic protocols; *AMI*, acute myocardial infarction; *BMI*, body mass index; *CT*, computed tomography; *eGFR*, estimated glomerular filtration rate; *ED*, emergency department; *hs-cTn*, high-sensitivity cardiac troponin; *IQR*, interquartile range; *NA*, not applicable; *NSTEMI*, non-ST-elevation myocardial infarction; *LDL*, low density lipoprotein.

*Hyperlipidaemia was defined by patients' records and/or active lipid-lowering treatment.

†n=1,054 for family history of atherosclerosis.

‡Not including initial triage. n=1,397 for time from ED arrival to evaluation by physician.

§12 patients excluded from risk group analysis due to missing 1-hour hs-cTn sample (9 in the POC group and 3 in the control group).

Table 2. Effectiveness outcomes.

Outcome	POC (n=728)	Control (n=766)	Difference (95% CI)
LOS in the ED, min, median (95% CI)	174 (167-181)	180 (175-189)	6 (−4 to 17)
LOS in the hospital, h, median (95% CI)	21 (19-22)	20 (18-21)	1 (−3 to 2)
Total LOS <3 h, n, % (95% CI)	115 16 (13-19)	113 15 (12-17)	1 (−5 to 2)
Total LOS <6 h, n, % (95% CI)	273 38 (34-41)	290 38 (35-41)	0 (−5 to 5)

POC, point of care; *LOS,* length of stay; *ED,* emergency department.

patients (8.2%, 95% CI 6.0 to 10.0) (point-of-care) and 60 out of 766 patients (7.8%, 95% CI 6.0 to 10.0) (control) were readmitted within 30-days.

LIMITATIONS

Several limitations should be considered. This single-center study only includes patients from Norway, which might limit generalizability. Patient characteristics and total rate of index NSTEMI (5.3%) resemble those in international studies, but ED processes and LOS may differ to other settings.[35,36] We enrolled patients from 07:00 AM to 10:00 PM, 7 days a week. The lack of enrollment during night shifts might affect generalizability, but the weekly time frame for inclusion is still similar to or larger than most comparable studies. Additionally, due to concurrent presentations, we were unable to include all patients. We

Table 3. Thirty days safety outcomes.

Total 30-D Safety				
Outcome	**POC (n=728)**	**Control (n=766)**	**Difference (95% CI)**	**Odds Ratio (95% CI)**
Composite death and AMI, n % (95% CI)	51 7.0 (5.2-8.9)	39 5.1 (3.5-6.7)	1.9 (−0.5 to 4.3)	1.4 (0.9 to −2.2)
Composite-AMI, acute revascularization, Death, n % (95% CI)	83 11.4 (9.1-13.7)	72 9.4 (7.4-11.5)	2.0 (−1.1 to 5.1)	1.2 (0.9 to −1.7)
AMI, n % (95% CI)	47 6.5 (4.7-8.3)	37 4.8 (3.3-6.4)	1.6 (−0.7 to 4.0)	1.4 (0.9 to −2.1)
Acute revascularization, n % (95% CI)	61 8.4 (6.0-10.0)	63 8.2 (6.0-10.0)	0.1 (−2.6 to 2.9)	1.0 (0.7 to −1.5)
Death, n % (95% CI)	4 0.5 (0.0-1.0)	4 0.5 (0.0-1.0)	0.0 (−0.7 to 0.8)	1.1 (0.3 to −4.2)
30-D safety after excluding index events				
	POC (n=728)	**Control (n=766)**	**Difference (95% CI)**	**Odds Ratio (95% CI)**
Composite death and AMI, n % (95% CI)	5 0.7 (0.1-1.3)	2 0.3 (−0.1 to 0.6)	0.4 (−0.3 to 1.1)	2.6 (0.5 to −13.6)
Composite-AMI, acute revascularization, death, n % (95% CI)	6 0.8 (0.2-1.5)	4 0.5 (0.0-1.0)	0.3 (−0.5 to 1.1)	1.6 (0.4 to −5.6)
AMI, n, % (95% CI)	3 0.4 (0.0-1.0)	1 0.1 (0.0-0.0)	0.3 (−0.2 to 0.8)	3.2 (0.3 to −30.5)
Acute revascularization, n % (95% CI)	2 0.3 (0.0-1.0)	3 0.4 (0.0-1.0)	0.1 (−0.5 to 0.7)	0.7 (0.1 to −4.2)
Death, n % (95% CI)	2 0.3 (0.0-1.0)	1 0.1 (0.0-0.0)	0.1 (−0.3 to 0.6)	2.1 (0.2 to −23.3)
30-D readmission, n % (95% CI)	60 8.2 (6.0-10.0)	60 7.8 (6.0-10.0)	0.4 (−2.4 to 3.2)	1.1 (0.7 to −1.5)

CI, confidence interval; *N/A,* not applicable.

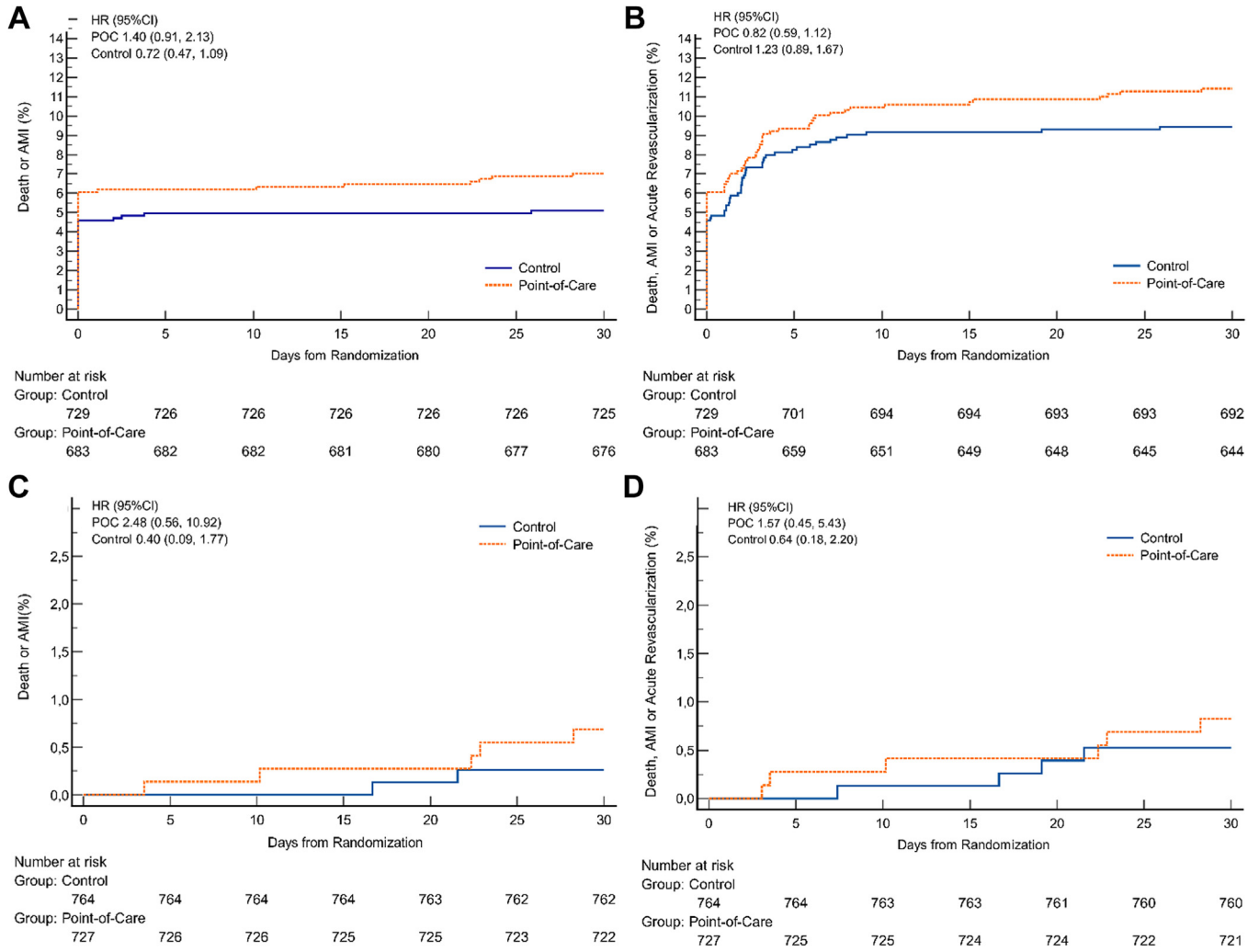

Figure 3. *A,* Kaplan-Meier curves for AMI and death from randomization to 30-day follow-up; *B*, AMI, acute revascularization and death from randomization to 30-day follow-up; *C*, AMI, and death from discharge to 30-day point; and *D*, AMI, acute revascularization and death from discharge to 30-day point.

estimate that approximately 6,000 patients with symptoms suggestive of acute coronary syndrome presented to the ED during the 24 months of inclusion, and our population represents 27% of them. Although we believe that our population is representative, some selection bias is possible.

We compared hs-cTnT with hs-cTnI in this study as we only had access to a central laboratory hs-cTnT assay at our site. Although cTnT and cTnI assays are considered to have the same diagnostic performance for NSTEMI, they are different biomarkers for which biological differences and analytical characteristics have been described.[43,44] We observed more patients diagnosed with index NSTEMI in the point-of-care group. This may be coincidental, but as opposed to the control group, admitted point-of-care patients would be tested using both troponin assays (hs-cTnI in the ED and hs-cTnT after admission), which may have led to a higher identification rate of NSTEMI.[45]

Furthermore, we observed a notable exclusion—4.5% of patients randomized to point-of-care—due to initial technical problems with the point-of-care assay. Differences in exclusion rates could introduce bias, and failure rates could also have led to increased workload and delayed diagnostic evaluation. In retrospect, registering failure rates throughout the study would have provided valuable insight.

Finally, in the point-of-care group, we also analyzed the admission hs-cTnT in the central laboratory, and clinicians had the option to make these available on special request if needed for comparison during the hospital stay, as any further hs-cTn sampling was done by the central laboratory hs-cTnT assay. The emergency physicians used this option in 27.9% of the point-of-care patients (Table 1). Unfortunately, we observed that they used it in 21.9% of the discharged point-of-care patients, and thus, not for

comparison during the hospital stay as intended. Whether this reflects distrust in the new point-of-care assay, unfamiliarity with a hs-cTnI assay, or other reasons is unknown, but it likely impacted the effectiveness of our evaluation.

DISCUSSION

To our knowledge, the WESTCOR point-of-care study is the first randomized clinical trial comparing point-of-care and central laboratory hs-cTn assays in the ED. It included 1,494 patients presenting to the ED with symptoms suggestive of acute coronary syndrome. The major strength of this study is the ability to report outcomes of an accelerated diagnostic protocols from point-of-care hs-cTn assay implemented and acted on in a real-life ED setting, eliminating some of the limitations attributed to earlier observational studies. In addition, to our knowledge, this is the first study to report on measurement of hs-cTnI in whole blood in real-time clinical use with measurements performed by intended users in the ED.

We found that point-of-care testing was associated with a 6-minute (95% CI −4 to 17) difference in median ED LOS, which was less than the minimum clinically meaningful difference of 15 minutes used in our power calculation. The prevalence of adverse events after discharge was low (<1%) in both groups, although the study was not powered for between-group comparison. Safety has been and will be further evaluated in earlier and ongoing studies like the Mersey Acute Coronary Syndrome Rule-Out trial 2 (ClinicalTrials.gov Identifier: NCT05322395).[35,36,38,46]

The potential for point-of-care testing to reduce ED LOS may have been limited by the relatively STAT for the hs-cTnT central laboratory assay at our hospital (71% to 91% of results reported in 60 minutes or more after sample arrival at the laboratory) and our ED LOS for patients with chest pain (approximately 3 hours) being lower than reported elsewhere.[16,18-20,22,23] Earlier studies have evaluated the reduction in LOS after introducing hs-cTn assays and/or accelerated diagnostic protocols in the ED, showing reductions in LOS varying from 6 minutes to several hours.[16-26] Studies with shorter baseline LOS reported smaller reductions.[21,24,25] Furthermore, we recommended an admission and 1-hour samples in all patients. Physicians were able to act on the admission sample, but we likely did not assess a single-sample rule-out strategy, potentially limiting effectiveness benefits.

The lack of a validated 0/1 algorithm for the point-of-care hs-cTnI assay also limited the potential benefit of point-of-care testing. The discrepancy in rule-out ability between the ESC accelerated diagnostic protocols and our suggested point-of-care hs-cTnI protocol (64% versus 39%) led to a much larger observation group (Table 1), indicating that the cutoffs used may not have been optimized for ED effectiveness. Previous studies have suggested a single-sample cutoff of less than 4ng/L to have a rule-out rate ranging from 19% to 44%.[36,47] Cullen et al[35] used a similar admission sample cutoff as our protocol in a 0/2 hour accelerated diagnostic protocols, achieving a rule-out rate of 53% to 64%. Differences in patient demographics and health care systems likely influence the algorithm's effectiveness. Moreover, shifts in assay stability may also affect performance, particularly rule-out ability.[8,48]

Clinical Implications of This Study

This study demonstrates that hs-cTn accelerated diagnostic protocols using point-of-care testing do not necessarily reduce ED LOS. Given the higher cost per analysis and the potential for increased workload for the ED staff associated with point-of-care testing, optimizing the clinical effectiveness is essential. Before implementing point-of-care testing, decisionmakers should consider other local logistical factors and potential barriers affecting ED patient throughput time.

In summary, implementing point-of-care hs-cTnI testing in the ED with a 0/1-hour diagnostic algorithm did not lead to a clinically meaningful reduction in ED LOS. The incidence of myocardial infarction, acute coronary revascularization, or death for 30 days of follow-up remained similar between the groups.

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Author affiliations: From the Emergency Care Clinic (Thulin, Jordalen, Lekven, Krishnapillai, Bjørneklett), Haukeland University Hospital, Bergen, Norway; Department of Heart Disease (Myrmel, Lekven, Krishnapillai, Steiro, Norekvål, Vikenes, Aakre), Haukeland University Hospital, Bergen, Norway; Division of Cardiovascular Sciences (Body), Faculty of Biology, the University of Manchester, Manchester, United Kingdom; Emergency Department (Body), Manchester University NHS Foundation Trust, Manchester, United Kingdom; St George's University of London (Collinson), London, United Kingdom; Department of Laboratory Medicine and Pathology (Apple), Hennepin Healthcare/HCMC, Minneapolis, MN; Department of Laboratory Medicine and Pathology (Apple), University of Minnesota, Minneapolis, MN; Department of Emergency Medicine (Cullen), Royal Brisbane and Women's Hospital, Brisbane, Australia; School of Medicine (Cullen), University of Queensland, Brisbane, Australia; Faculty of Health (Cullen), Queensland University of Technology, Brisbane, Australia;

Department of Clinical Science (Norekvål, Vikenes, Aakre), University of Bergen, Bergen, Norway; Health Services Research Unit (Norekvål, Wisløff), Akershus University Hospital, Lørenskog, Norway; Institute of Clinical Medicine (Wisløff, Omland), University of Oslo, Oslo, Norway; Department of Clinical Medicine (Bjørneklett), University of Bergen, Bergen, Norway; Department of Cardiology (Omland), Akershus University Hospital, Lørenskog, Norway; and Department of Medical biochemistry and Pharmacology (Aakre), Haukeland University Hospital, Bergen, Norway.

Author contributions: KMA conceived the study, designed the trial, obtained research funding, and supervised the conduct of the trial. IVLT supervised recruitment of participants, established the Case Report Firm, collected and plotted the data. IVLT and GMSM performed the statistical analysis. KMA, KV, and ROB were included in the steering committee. TO, POC, FSA, LC, and RB were scientific advisers for the study and critically reviewed and revised the manuscript. OCL, JK, SMFJ, OTS, and TMN provided scientific advice during the conduction of the study. OCL and JK performed adjudication of index diagnosis. TW provided statistical advice on study design. IVLT drafted the manuscript, and all authors contributed substantially to its revision. KMA takes responsibility for the paper as a whole.

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