Improving Care with the First measurement of high-sensitivity troponin T (ICare-FirsT) to enable early rule out and reduce length of stay: a diagnostic and observational study

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ABSTRACT

Background Pathways incorporating clinical risk assessment, ECG and serial troponin measurements for the assessment of patients with possible myocardial infarction (MI) in the ED are standard practice. Incorporating a single troponin test to stratify to low risk of MI using a baseline measurement of cardiac troponin (cTn) with a high-sensitivity T assay (hs-cTnT) is recommended. We aimed to implement a pathway incorporating a single-test component and measure the impact on length of stay (LOS).

Methods There were two study phases: (1) Development and performance assessment of a novel pathway incorporating a single-test hs-cTnT stratification using high-fidelity research data, (2) An audit of the implementation of a single-test Roche hs-cTnT strategy within multiple EDs. The low-risk threshold used for hs-cTnT was 5 ng/L. The safety metric was MI or death not known to be non-cardiac within 30 days (MACE30).

Results Phase I: The derived pathway had 16.3% low risk after one blood draw ≥3 hours from symptom onset with hs-cTnT <5 ng/L, non-ischaemic ECG and ED Assessment of Chest pain Score <21.
Phase II: In six hospitals, there were 10 912 patients in the control arm and 13 997 after implementation of single-test hs-cTnT. The unadjusted estimated mean reduction in LOS after intervention was 1.6% (95% CI 0.4% to 2.9%). After adjustment accounting for increased presentations, this was 8.5% (95% CI 7.7% to 9.3%).

Conclusions Within clinical pathways, a single test with a result from an hs-cTnT of <5 ng/L as a component resulted in a small, but meaningful, reduction in mean ED LOS.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ High-sensitivity cardiac troponin (hs-cTn) assays have the potential to screen out patients at low risk of myocardial infarction with one low result.

WHAT THIS STUDY ADDS

⇒ Real-world experience and performance metrics, particularly ED length of stay (LOS), after implementing within existing accelerated diagnostic pathways (ADPs) a single blood test component with the Roche hs-cTnT assay across multiple hospitals.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ That within ADPs, a single-test risk stratification with hs-cTnT is feasible and safe, and reduced lengths of stay.
⇒ The extent of reduced LOS likely depends on other factors, not all of which are easily accounted for and should be considered when looking to change practice.

INTRODUCTION

Accelerated diagnostic pathways (ADPs) which combine history, risk factors and ECG findings with troponin concentrations have been shown to safely reduce length of stay (LOS) and improve consistency of practice.[1 2] They allow for the rapid identification and discharge of those patients with a low risk of Acute Myocardial Infarction (AMI), while ensuring that those who are high-risk are prioritised for expedited ongoing assessment and management.

During 2015, New Zealand became the first country to adopt ADPs for the assessment of possible acute coronary syndrome (ACS) across all EDs.[3] At the time, the evidence for incorporating within ADPs a single-test cTn measured with a high-sensitivity cardiac troponin (hs-cTn) assay result to screen for low-risk stratification (rule-out) was preliminary, and none of the hospitals adopted this approach. Later large meta-analyses provided strong evidence to support the creation of rapid discharge pathways for patients with a single very low initial troponin if presenting >2–3 hours after pain onset.[4 5] The meta-analysis of the hs-cTnT assay, relevant to this study, of 9241 patients from 11 cohorts across multiple countries found troponin concentrations below the limit of detection (LoD) classified 30.6% as low risk.[4] The sensitivity was high, 98.7%, and of the 14 false negatives, 7 were <3 hours from symptom onset. Subsequently, two approaches to the use of hs-cTn have emerged which recommend low-risk stratification for non-early presenters using a single hs-cTn measurement: (1) In hs-cTn only pathways appearing in the European Society of Cardiology (ESC) guidelines with the caveat that troponin measurements were

to be 'used in conjunction with clinical and ECG findings'[6] and (2) In pathways containing a formal risk score, ECG findings, and hs-cTn.[2 7 8] Previously, we reported on this latter strategy as Christchurch Hospital became the first to adopt and report on a single hs-cTn (Abbott ARCHITECT high-sensitivity I assay, hs-cTnI) measurement for early rule-out of ACS within an ADP.[9] Prior to these studies, an audit of current hs-cTnT use at multiple hospitals had been conducted.

The expectation was that the introduction of a single blood sample low-risk stratification strategy using hs-cTnT within existing ADPs would safely reduce mean LOS for patients discharged home from ED. Our primary objective was to safely implement a single test rule-out within current ADPs in hospitals using hs-cTnT, namely: on the first blood sample to screen patients presenting more than 2–3 hours post symptom onset for eligibility for early discharge because of a very low hs-cTnT result.

METHODS

This implementation evaluation comprises two studies: First, development and performance assessment of a novel ADP incorporating a single hs-cTnT stratification using high-fidelity research data (phase I). Second, audit of the implementation of a single hs-cTnT strategy within multiple New Zealand EDs (phase II). Both phases used the Roche Diagnostics fifth generation hs-cTnT immunoassay with a limit of blank of 3 ng/L, LoD of 5 ng/L, limit of quantitation of 6 ng/L, and upper reference level of 14 ng/L (Cobas E411 analyser).[10]

Patient and public involvement

Patients were not involved in the development of research questions or protocols, but public lay members are on all New Zealand ethics committees and consultation with Māori representatives is undertaken prior to completion of protocols. In phase I patients were recruited and consented for extra blood samples in the ED. For phase II, all data were collected retrospectively from clinical records. Results of these and other of our studies are regularly disseminated to public groups through public presentations.

Inclusion and exclusion

All patients ≥18 years of age who attended a study ED and received at least one hs-cTnT test were included. The primary safety metric was sensitivity, and secondary negative predictive value (NPV), for AMI or death unless clearly non-cardiac (Major Adverse Cardiac Event, MACE) within 30 days of ED presentation (MACE30). Within New Zealand, all people who have ever encountered the health system have a unique identifier (National Health Index, NHI). The NHI links all ED presentations, admissions, blood results and mortality events. We used this linkage to extract information from hospital electronic health records, the New Zealand national death registry on all events within 30 days for all patients. Ethnicity is self-selected as the group people identify with. Sex data are recorded as Male, Female, Unknow, or Indeterminate.

Demographic data are presented as mean±SD, median (lower quartile to upper quartile), or n (%) as appropriate. Diagnostic metrics are presented with 95% CIs. All data analysis used R version 4.3.[11]

Phase I: Development and internal validation of a novel ADP

We aimed to establish an ADP[12 13] incorporating a risk assessment score (ED Assessment of Chest pain Score (EDACS) or clinician gestalt), ECG findings, and troponin (hs-cTnT assay) with an initial low-risk stratification step when baseline troponin was <5 ng/L.[4] Patients were prospectively recruited in three periods with aligned recruitment methodology: 11 October 2010 to 14 July 2012, 26 June 2013 to 30 July 2014, and 5 July 2016 to 5 January 2018 as part of two randomised controlled studies of ADPs and three observational studies from our ongoing ED recruitment (ACTRN12611001076965).[2 14–18]

Participants were consented adults ≥18 years presenting acutely to a tertiary metropolitan ED (Christchurch) with symptoms suggestive of myocardial infarction (MI) in whom the attending physician(s) planned to investigate with serial cardiac troponin measurements. The principal exclusion criteria were ST-segment Elevation MI (STEMI), or a clear non-cardiac cause for symptoms, ongoing chest pain, haemodynamic instability or crescendo angina. Additional blood samples were stored at –80°C in order to perform assessments of future tests such as described here. Details of ethics approval for the data collected are in the original papers[2 14–18], in all cases patients provided written consent.

The primary outcome was an ADP incorporating a single baseline hs-cTnT <5 ng/L low-risk stratification strategy. The ADP performance was assessed by the sensitivity for MACE30 of the low-risk branch of the pathway and the proportions of patients in each branch. MACE30 was adjudicated using the clinical troponin (Abbott contemporary cTnI or hs-cTnI) by two clinicians blinded to each other, and with a third cardiologist used to settle any conflicting diagnoses.[2 14–16 18] MI was classified based on the global task force's universal definition that was in place at the time of each study,[19 20] which required clinical evidence of myocardial ischaemia (ischaemia symptoms, ECG changes or imaging evidence) along with evidence of necrosis based on a rising or falling pattern of troponin (a delta of ≥20% was used), with at least one concentration above the troponin 99th percentile and blood draws at least on arrival in the ED and 6–12 hours later. Internal validation employing bootstrapping with 500 bootstrapped samples was used to determine the 95% CI for each metric.

Phase II: Monitored implementation into clinical practice

We aimed to implement into clinical practice in 11 hospitals ADPs which incorporated stratification to low risk following a single troponin measurement. The secondary aim was to measure the safety and effectiveness of the change practice. Six hospitals agreed to participate in data collection, and their ADPs are presented in online supplemental file 1. Complete data were obtained retrospectively from five hospitals. Data collection was from 1 September 2018 to 31 August 2022. The intervention at each hospital started between March 2019 and March 2021 (online supplemental figure 2.1).

Participants were adults ≥18 years in hospitals that used hs-cTnT and changed from an ADP incorporating only two troponin measurements to one that allowed for the early risk stratification in patients with a single baseline hs-cTnT <5 ng/L. Support for each hospital was provided by the study team to adjust ADP documentation.

Each hospital had a minimum 6-month control period prior to the intervention phase. Usual care (the control arm) is defined as the 'existing daily practice (existing ADP) of the attending clinical staff to diagnose a patient with chest pain'. These pathways all incorporate a risk score, with one exception where clinician gestalt was used as part of an ESC guidelines style pathway,[6] an ECG and two central laboratory troponin measurements with

hs-cTnT. While the advice generated by the troponin results, risk scores and ECG is consistent, each hospital has tailored pathway components around such actions as admission and secondary testing. All laboratories reported any presence of haemolysis to clinicians.

The intervention was a modification to existing ADPs which introduced a new, very early low-risk stratification path for patients based on a single troponin measurement with hs-cTnT on a blood sample drawn on arrival to the ED ('baseline'). This result may be used to 'screen-out' patients not requiring further troponin measurement. This was the only change to ADPs, and the use of ECG and EDACS was not modified. Where patients do not meet the threshold for baseline low-risk stratification using hs-cTnT, ECG or EDACS, this will trigger for them to continue to follow the rest of the unchanged usual care pathway (ie, a second blood sample will be drawn at 2–3 hours for a second troponin measurement). Data were collected for the intervention period of at least 4 months at each hospital.

Only the first presentation of each patient was used for the primary analysis. As a pragmatic before-after study using electronic health record data collected retrospectively, we were unable to access ECG or EDACS results and therefore had to consider all patients receiving a troponin measurement in the ED. The primary outcome was ED LOS. Secondary outcomes were the proportions with LoS <2 hours, <4 hours and <6 hours. Subgroup analysis was by hospital, ethnicity and sex. The safety outcome was the rate of MACE30. MACE30 was determined by International statistical Classification of Disease and related health problems revision 10 (ICD10) codes for STEMI (I21.0 or I21.1 or I21.2 or I21.3) and Non-STEMI (NSTEMI) (I21.4 or I21.9 or I22.0 or I22.1 or I22.8 or I22.9).

This phase was a pragmatic prospective implementation study with a retrospective multisite before-after data analysis. As a pragmatic study, the date for modified ADP implementation at each hospital was planned around wider ED workflows. Given this, and that over the period of data collection the EDs became busier with more presentations of patients, including those with COVID-19, we used a regression model to estimate the influence of the intervention on LOS, namely $\log_2$(ED LOS) ~ data collection period + time + season + shift + time from presentation to first blood draw, where the reference value for data collection period was the control arm, time was number of months from the beginning of the data collection, season accounted for known variations in presentations in each season, and shift was one of three shifts occurring each day in ED, and time from presentation to first blood draw is a variable used to account for ED business. Time from presentation to first blood draw was included after simulation on a dummy data set to see if it could help account for increased business. The outcome variable was log transformed as this was found in simulations to reduce residuals (better quantile-quantile, QQ, plots). For data analysis purposes only there was a 'run-in' period of 2 months at each hospital from the start of the use of the modified ADP (the intervention). The intervention period for analysis began at the end of the 2 months. This was because it is known that it can take some time for interventions, and their effect, to become embedded.[2]

The Health and Disabilities Ethics Committee confirmed that this observational study was out of scope, and therefore did not require additional ethical review.

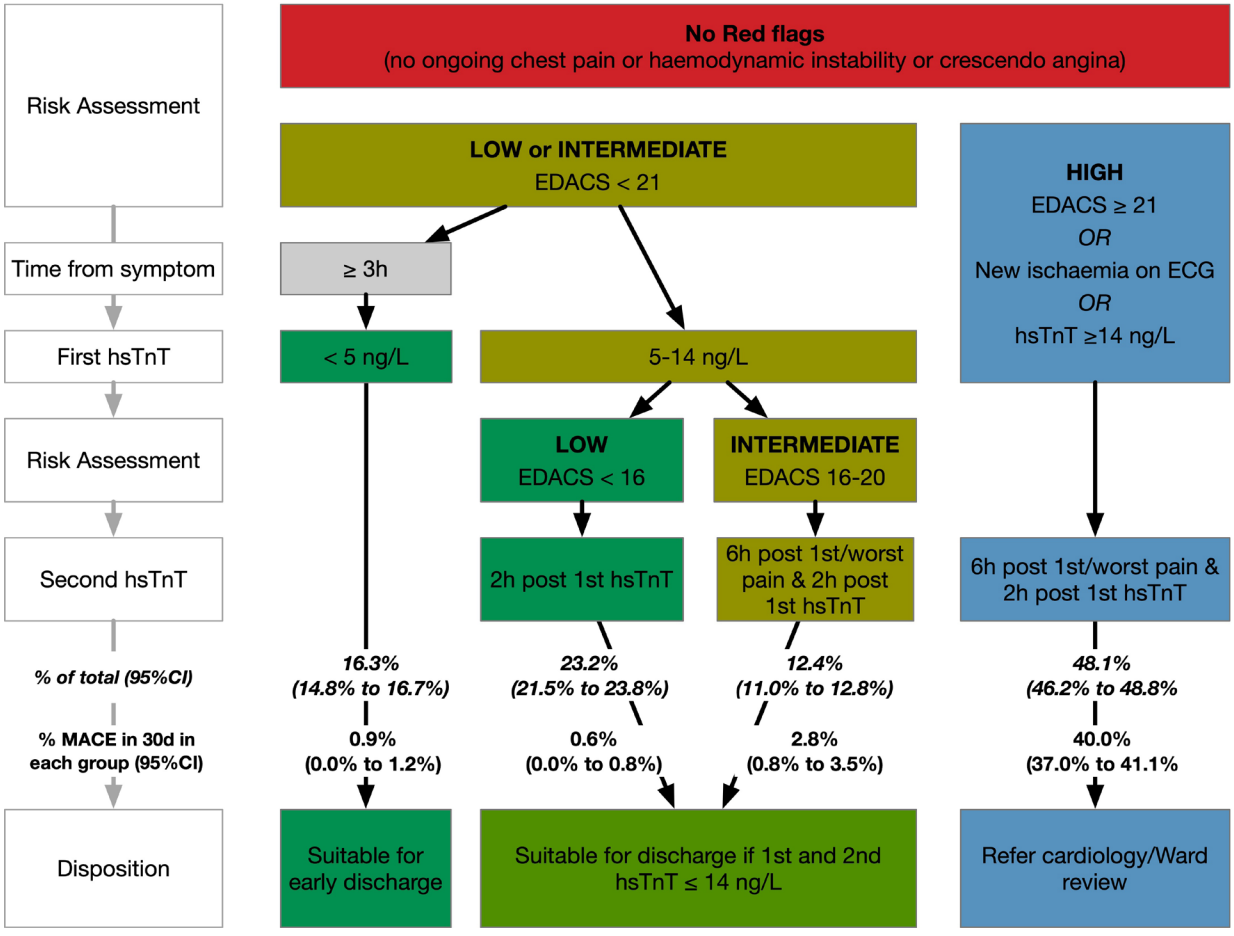

Figure 1 Developed and internally validated pathway (study phase I). EDACS, ED Assessment of Chest pain Score.

RESULTS

Phase I

There were 2050 patients ≥18 years of whom 407 (19.9%) had MACE30. MACE30 patients were 6 years older on average, and more likely to be male than patients without a MACE30 (75.2% cf 60.2%) (online supplemental table 1.1).

There were 331 (16.3%) patients with baseline troponin <5 ng/L, no new ischaemia on ECG, ECACS <21, with blood sample ≥3 hours from symptom onset who were eligible for early discharge, figure 1. The sensitivity was 99.3% (95%CI: 97.9% to 99.8%). The 95% CI of the proportion <5 ng/L was 14.8% to 16.7% and the proportion <5 ng/L with a MACE30 was 0.9% (95%CI 0.0% to 1.2%).

48.1% (46.2% to 48.8%) of patients were high-risk (EDACS≥21 or new ischaemia on ECG or at least one of two troponin ≥14 ng/L), of whom 40.0% (37.0% to 41.1%) had a MACE30, figure 1.

Phase II

Eleven New Zealand hospitals have now implemented an ADP including low-risk stratification with hs-cTnT <5 ng/L, ECG, and EDACS. Two others used <5 ng/L within a European Society of Cardiology guideline pathway.[6] 77 187 presentations met the inclusion criteria, of which 40 810 were excluded (Troponin >90 min after presentation, 19 287; ED LOS>12 hours 9960; STEMI, 921; Died in ED, 57; Admitted after only one troponin, 10 585). A further 5689 presentations were excluded as second or subsequent presentations for the same patient. Finally, 1612 patients were in the run-in phase, leaving 10 912 in the control and 13 977 in the intervention arms. Patients appeared younger and more likely to be female in the intervention arm, table 1. The MACE30 rate was lower in the intervention arm.

Following implementation the median ED LOS (IQR) changed from 4.92 hours (3.65–66.32 hours) in the control period to 4.85 hours (3.37 to 6.63 hours) in the intervention phase, online supplemental figure 2.2. The unadjusted estimated mean reduction in ED LOS was 1.6% (95% CI 0.4% to 2.9%). In a multivariable analysis (table 2), including accounting for time from start of data collection and season, there was an estimated 8.6% (95% CI 7.1% to 10.0%) reduction in ED LOS. For a patient presenting in the morning shift in winter with a blood draw 0.5 hours after ED presentation, the effect of the intervention was a reduction in ED LOS of 29 min. Table 4 illustrates other scenarios where season, shift, and ED presentation time were randomly chosen. The mean 8.6% reduction corresponds to an estimated mean 27 min reduction in LOS.

The non-adjusted proportions leaving the ED in <2 hours, and <4 hours were greater after the ADP change than before, but the proportion <6 hours was slightly less. However, the adjusted analysis showed that the odds for leaving the ED in <6 hours was marginally greater after the change of practice than before, table 4.

Table 1 Demographics (Study Phase II)

	Control (n=10 912)	Intervention (n=13 997)	Total (n=24 169)
Age (years)			
Mean (SD)	63.5 (16.5)	58.2 (17.6)	60.4 (17.3)
Range	18–102	18–102	18–102
Sex n (%)			
Female	5268 (44.2%)	8217 (46.9%)	13 485 (45.8%)
Male	6640 (55.8%)	9319 (53.1%)	15 959 (54.2%)
Unknown	1 (0.0%)	1 (0.0%)	2 (0.0%)
Ethnicity n(%)			
Māori	2014 (16.9%)	2495 (14.2%)	4509 (15.3%)
Pacific Peoples	1191 (10.0%)	3505 (20.0%)	4696 (15.9%)
Non Māori/non Pacific Peoples	8699 (73.0%)	11 535 (65.8%)	20 234 (68.7%)
Unknown	5 (0.0%)	2 (0.0%)	7 (0.0%)
Triage level n(%)			
Missed	5	1	6
1	86 (0.7%)	184 (1.0%)	270 (0.9%)
2	7844 (65.9%)	10 060 (57.4%)	17 904 (60.8%)
3	3722 (31.3%)	6623 (37.8%)	10 345 (35.1%)
4	246 (2.1%)	653 (3.7%)	899 (3.1%)
5	6 (0.1%)	16 (0.1%)	22 (0.1%)
MACE30	693 (6.3%)	711 (5.1%)	1404 (5.8%)

Table 2 Adjusted analysis: Model log2(ED LOS) ~ Data collection period+time + season+shift (Study Phase II)

Independent variable	β estimate (SE)	t-value	P value
UNADJUSTED ANALYSIS			
Data collection period			
Control	Reference		
Intervention	−0.023 (0.009)	−2.6-	0.01
ADJUSTED ANALYSIS			
Data collection period			
Control	Reference		
Intervention	−0.129 (0.012)	−10.9	<0.0001
Time (months) from data collection start	0.0081 (0.0006)	14.1	<0.0001
Season			
Autumn	Reference		
Winter	0.060 (0.013)	4.7	<0.0001
Spring	0.060 (0.014)	4.4	<0.0001
Summer	0.022 (0.013)	1.7	0.10
Shift			
Morning	Reference		
Afternoon	−0.094 (0.011)	−8.8	<0.0001
Evening	0.081 (0.011)	7.3	<0.0001
Time from presentation to first blood draw	0.03 (0.01)	2.1	0.03

Table 3 Examples of the difference by the end of the study period (study phase II)

Season	Shift	Presentation to first blood sample (h)	Difference (mins)
Autumn	Morning	0.75	27.8
Autumn	Afternoon	0.75	26
Winter	Morning	0.25	28.7
Winter	Morning	1.5	29.4
Spring	Morning	0.75	29
Spring	Morning	2	29.7
Spring	Afternoon	0.75	27.2
Spring	Afternoon	0.75	27.2
Spring	Evening	1	30.9
Summer	Afternoon	0.5	26.3
Summer	Afternoon	4	28.3
Summer	Evening	0.5	29.7

LOS, length of stay.

Table 4 Proportions discharged from ED within set time periods (Study Phase II)

Time period	Univariate proportions (%)		Adjusted OR (95% CI)
	Control	Intervention	
< 2 hours	3.6%	5.5%	2.9 (2.5 to 3.4)
< 4 hours	32.0%	35.8%	1.6 (1.5 to 1.7)
< 6 hours	69.7%	66.6%	1.0 (1.0 to 1.1)

For males and females, and for Māori and Non-Māori/non-Pacific peoples the intervention reduced LOS, but this was unable to be demonstrated for Pacific Peoples, online supplemental tables 3.2 and 3.3.

DISCUSSION

Two phases of the study were conducted as lead into and implementation of the use of hs-cTnT to risk stratify patients with possible AMI after a single blood draw in all New Zealand hospitals that use the Roche hs-cTnT assay.

Phase I used a high-fidelity research dataset to explore the baseline low-risk approach further and in conjunction with ECG and EDACS, by developing a prototype pathway and calculating projected event rates for each pathway stream and likely proportions of patients allocated to each pathway stream.

The pathway prototype suggested that such an approach is plausible and that the baseline low-risk at screening is applicable to 20% of patients with a 30-day event rate for MACE30 at 0.9%, which was considered acceptable.[21]

We note the ESC Guidelines use a delta of ≥4 ng/L between 0 and 2 hours troponins as a trigger for additional testing. This may be added to the middle stream of low-intermediate EDACS, no new ischaemia, and two hs-cTnT ≤14 ng/L. 0.6% of all patients met this criterion.

For phase II existing ADPs were adjusted to include low-risk stratification of some patients with hs-cTnT <5 ng/L on presentation to the ED. This resulted in shorter lengths of ED stay, with more patients being discharged from ED within 4 hours. The unadjusted reduction in LOS was modest, most likely because during the period all New Zealand EDs faced increasing rates of presentation to the ED without commensurate increases in staffing resulting in delays to discharge. The adjusted analysis, which attempted to account for these non-intervention related forces which increase LOS, demonstrated the likely impact of the intervention was that it reduced length of stay by 27 min. Within the New Zealand health system, there are tens of thousands of patients per annum assessed with the Roche troponin assay, meaning implementation of a single-test component of the pathway can save tens of thousands of hours of patient and staff time.

It is possible that there was an increased use of troponin tests in lower risk patients given the lower mean age and lower MI rate in the intervention group. This may reflect the use of troponin in patients suspected of myocarditis, a practice which became more common with the introduction of COVID-19 vaccines part way through the data collection period. As with patients being investigated for possible MI, those being investigated for possible myocarditis would also benefit from the more rapid evaluation with a single blood draw.

Previous work has demonstrated the safe use of a single troponin measurement with an hs-cTnI assay within a clinical pathway. We first demonstrated this at a single hospital.[9] Far more substantially, the large stepped-wedge cluster randomised controlled trial in Scotland, high-sensitivity cardiac troponin on presentation to rule out myocardial infarction (HiSTORIC) demonstrated a safe reduction in length of ED stay and a higher proportion of patients discharged with a pathway incorporating only hs-cTnI and for those without diagnostic ECG changes compared with usual practice without a single-sample low-risk stratification step.[22] There has been little shown with hs-cTnT. Carlton *et al* concluded in a randomised controlled trial of 629 participants that the introduction of an ADP incorporating a single sample troponin measured with a hs-cTn assay facilitated "safe early discharge in >40% of patients with chest pain".[23] The adjusted odds ratio for discharge without 30d MACE within 4 hours for the single-sample strategy compared with usual care was 1.58 (95% CI 0.84 to 2.98). Of the eight participating hospitals, five employed hs-cTnT. Half the hospitals already had a single-sample low-risk discharge procedure in conjunction with a risk score (History, ECG, Age, Risk factors and Troponin, HEART, in each case). In the intervention, patients were eligible for discharge if 'their ECG was non-ischaemic; a single hs-cTn test taken at presentation (and irrespective of the symptom onset time) was undetectable … and there was no ongoing clinical concern'. The Troponin-only Manchester Acute Coronary Syndromes approach also uses only one hs-cTnT result along with the ECG and several symptoms in a model that produces a probability of ACS showed great potential to safely low-risk stratify patients in its validation cohort.[1] Similarly, the Artificial intelligence in suspected MI study also produced risk predictions and demonstrated good performance metrics with a single measurement with the hs-cTnT assay.[24] The post-implementation performance of these two risk prediction models has yet to be reported.

The strength of this current study was the utilisation of two approaches to assess the potential impact of a single sample low-risk stratification step with the hs-cTnT assay within an ADP. There are, though, several limitations. We were unable to randomise the implementation dates of the hospitals in the implementation study, nor were all 13 hospitals able to provide data for this analysis. Because of the large numbers involved we had to rely on electronic recording, and this meant we could not report on ECG findings or risk scores. Therefore, the proportions with a first troponin ≤5 ng/L are likely an overestimate of the numbers who were actually considered low-risk. Additionally, we relied on ICD10 coding for the diagnoses. Previously, we have shown in one New Zealand hospital that this has very good, but not perfect, correlation with double adjudication by senior physicians.[3]

We have demonstrated that a real-world change from ADPs requiring two troponin T measurements to ADPs with a possibility of utilising only one measurement for some patients did result in a reduction in ED LOS. However, it was a modest reduction, and perhaps less than what may have been expected from a retrospective study of the proportion of patients with hs-cTnT <5 ng/L and an ADP derived with a high-fidelity research database. Other factors at play during implementation, such as increased presentation to ED and use of testing on new patient groups, need to be considered and recognised as possibly influencing the effectiveness of the new strategy.

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Contributors Data collection: JWP, MPT, CP, AMR, RT. Design: phase I: MPT, CP, AMR, RT, JWP; phase II: GD, MPT. Statistical Analyses: JWP. Interpretation: JWP, LJ, JWP. Manuscript draft: JWP, LJ, JWP. Final approval: all authors. JWP is the guarantor, accepts full responsibility for the finished work and the conduct of thestudy, had access to the data, and controlled the decision to publish.

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Competing interests JWP has since the completion of the analysis engaged in a consulting agreement with Roche Diagnostics and has undertaken statistical consultancy for Siemens Healthineers, Radiometer, Abbott, QuidelOrtho and Upstream Medical Technologies. MPT has received honoraria, consultancy fees, and research funding from Abbott, Alere, Beckman, QuidelOrtho, Radiometer, Roche and Siemens. AMR holds an advisory board position with Roche Diagnostics, has received research grants from Roche Diagnostics and Novo Nordisk, and has received support in kind (immunoassay costs) from Roche Diagnostics and Sphingotec. RT holds an advisory board position and has received research grants from Roche Diagnostics, and has consulted and received research grants from Merck, Bayer and American Regent. CP is an employee and shareholder of Upstream Medical Technologies (which had no part in the design, funding or conduct of this study), has a services agreement with Radiometer ApS and has received support in kind and research funding from Biovendor CZ, Astra Zeneca, Roche Diagnostics and Radiometer ApS.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Consent obtained directly from patient(s)

Ethics approval This study involves human participants. Phase I: Details of ethics approval for the data collected are in the original papers[2 14–18], in all cases patients provided written consent. Phase II: The Health and Disabilities Ethics Committee confirmed that this observational study was out of scope, and therefore did not require additional ethical review. Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request. Phase I: Data may be requested from the lead author and may be made available under specific terms. Phase II: Data are from routinely collected health data and are not available.

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