In this issue: Measurably better healthcare

The Journal of the International Federation of Clinical Chemistry and Laboratory Medicine

eJIFCC

Reducing patient risk and enhancing care through the development and implementation of a new chest pain pathway, expedited by and for the COVID-19 era

Martin P. Than[1], John W. Pickering[1,2], Philip Adamson[4], Thomas Clendon[4], Christopher M. Florkowski[3], John Lainchbury[4], Jacques Loubser[1], Alison Nankivel[4], Sally J. Aldous[4]

[1] *Department of Emergency Medicine, Christchurch Hospital, Christchurch, New Zealand*
[2] *Department of Medicine, University of Otago Christchurch, Christchurch, New Zealand*
[3] *Canterbury Health Laboratories, Christchurch Hospital, Christchurch, New Zealand*
[4] *Cardiology Department, Christchurch Hospital, Christchurch, New Zealand*
[*] *On behalf of the Christchurch COVID pandemic chest pain pathway initiative members and wider stakeholder group*

ARTICLE INFO

Corresponding author:
Dr. Martin Than
21 Taylors Mistake Road
Sumner, Christchurch 8081
New Zealand
E-mail: martinthan@xtra.co.nz

Key words:
high-sensitivity cardiac troponin, acute myocardial infarction, emergency department, emergency room, accelerated diagnostic pathway, prognosis, coronavirus, COVID-19

ABSTRACT

The COVID-19 pandemic raised major concerns relating to hospital capacity and cross-infection patients and staff in the Emergency Department (ED) of a metropolitan hospital servicing a population of ~500,000. We determined to reduce length of stay and admissions in patients presenting with symptoms of possible myocardial infarction; the most common presentation group.

After establishing stakeholder consensus, the existing accelerated diagnostic pathway (ADP) based on the ED Assessment of Chest-pain Score (EDACS), electrocardiogram, and troponin measurements with a high-sensitivity assay (hs-cTn) on presentation and two hours later (EDACS-ADP) was modified to stream

patients following an initial troponin measure as follows: (i) to a very-low risk group who could be discharged home without follow-up or further testing, and (ii) to a low-risk group who could be discharged with next-day follow-up community troponin testing. Simulations were run in an extensive research database to determine appropriate hs-cTnI and EDACS thresholds for risk classification. This COVID-ADP was developed in ~2-weeks and was implemented in the ED within a further 3-weeks.

A comparison of all chest pain presentations for the 3 months prior to implementation of the COVID-ADP to 3 months following implementation showed that there was a 64.7% increase in patients having only one troponin test in the ED, a 30-minute reduction of mean length of stay of people discharged home from the ED, and a 24.3% reduction in hospital admissions of patients ultimately diagnosed with non-cardiac chest pain.

❖❖❖❖❖

INTRODUCTION

On 23 March 2020, New Zealand entered a stringent lockdown because of rising Sars-Cov2 cases nationally. Hospitals and their Emergency departments (EDs) began preparations for an influx of cases. Major concerns included cross-infection of patients and staff in the ED and bed space availability. Patients presenting with symptoms of chest pain and possible acute myocardial infarction (AMI) are one of the more common presentation groups to the ED accounting for approximately 5-15% of all presentations (1,2). We recognised that being able to reduce the length of stay in the ED of this patient group by expediting discharge of low-risk patients could reduce the risk of cross-infection and free up staff for dealing with more serious illnesses.

Christchurch Hospital has been a prominent developer of Chest Pain pathways. This has included running the world's first randomised controlled trial (RCT) evaluating a structured clinical pathway against usual care (3), and then the development of and subsequent validation in a second RCT of the Emergency Department Chest pain Score (EDACS) and pathway (4). In 2019 a modification of that pathway was implemented that used a troponin threshold of < 5ng/L (Abbott Architect – high sensitivity assay) to rule-out AMI after a single troponin measurement (5). This modification was based on a considerable body of evidence, which included locally collected data, that showed such a threshold safe and effective (6,7).The clinical approach at the time adopted a 'next day' community troponin test for patients discharged from the ED after a single troponin test.

The successive research studies had resulted in the creation of a large high-fidelity data set of laboratory and clinical variables for patients who had been assessed for possible AMI. We aimed to use this data to modify the existing pathway and to implement an expedited change of practice to reduce time spent in ED amongst these patients.

MATERIAL AND METHODS

This study measured the impact of a change practice, namely the implementation of a modified chest pain accelerated diagnostic pathway in the COVID era (the COVID-ADP). We present data from 3-months prior to the change of practice to 3-months post the change of practice. We will describe (a) pathway development, (b) pathway evaluation methods and (c) change management processes.

Pathway development

The COVID-ADP was developed by using a well characterised and high-fidelity data set of

patients who had presented to the Emergency Department at Christchurch Hospital with symptoms suggestive of AMI and in whom the attending physician intended to investigate for possible AMI.

This data set comprised four research studies during which patients were recruited with almost identical exclusion criteria which have been reported in detail elsewhere (3,4,8,9). Briefly, patients were excluded if <18 years, unable or unwilling to provide consent, a clear cause of symptoms other than AMI, ST segment Elevated Myocardial Infarction (STEMI), transfer from another hospital, pregnancy, unable to be followed-up, or staff considered recruitment inappropriate (e.g. receiving palliative care).

In all these studies participants had serial blood samples taken at the time of recruitment shortly after presentation (0h) and two hours later (2h). Blood was drawn into lithium-heparin tubes, immediately centrifuged, and stored at −80°C for later testing. Troponin concentrations were measured with a high-sensitivity Troponin I (hs-TnI) assay (Abbott ARCHITECT i2000 (Abbott Diagnostics, Chicago, Illinois).

The clinical troponin assay in use during the six-month quality improvement study was the same Abbott hs-TnI assay. Prior to implementation of the COVID-ADP all patients attending the ED with symptoms suggestive of AMI were assessed using the Emergency Department Assessment of Chest pain Score (EDACS) accelerated diagnostic pathway inclusive of a single troponin early rule-out (Figure 1).

We used the same definition of Major Adverse Cardiac Events (MACE) as with previous studies, either an AMI, cardiogenic shock, cardiac arrest, emergency revascularisation, ventricular arrhythmia requiring intervention, high-degree atrioventricular block needing intervention or death (unless clearly non-cardiac).

The Abbott ARCHITECT hs-cTnI assay has a limit of detection of 1.9 ng/L, and 99th percentile upper-reference-limits (URL) of 16 ng/L for Females and 34 ng/L for Males and an overall URL of 26 ng/L.

A priori we aimed to:

1. Identify an hs-cTnI threshold for use with a single troponin measure below which <1% of patients had a 30-d MACE,
2. Identify a group of low-risk patients where a second troponin measurement could be performed in the community. By consensus this group was to have approximately ≤ 5% 30-d MACE,
3. Determine a minimum time threshold from symptom onset for early presenters before which required an ongoing observation period and serial cTn measurements,
4. Specify a change in troponin concentration threshold which would trigger a review by a Cardiologist in patients receiving a next-day community troponin measurement, and
5. Design the pathway to reduce duplication of assessment of patients by ED and Cardiology doctors.

We used a process of iterating troponin thresholds to determine thresholds which produced the necessary metrics.

Preference was given to using the pre-existing troponin threshold of 5 ng/L for single sample rule-out (aim 1) as this was already in use and has been well validated internationally (5,6). To aid clinical acceptability we determined to utilise one or more of the risk thresholds for EDACS already in use at Christchurch Hospital, namely EDACS <16 for low-risk, 16-20 for intermediate risk and ≥21 for high-risk (5). A consensus decision was made *a priori* that evidence of new ischaemia on an electrocardiogram would remain being defined as a high-risk feature.

Since staff members were unable to meet face-to-face except for direct patient care (during the pandemic lockdown), iterative changes to the pathway format were achieved using consensus achieved via successive videoconference meetings.

Pathway evaluation

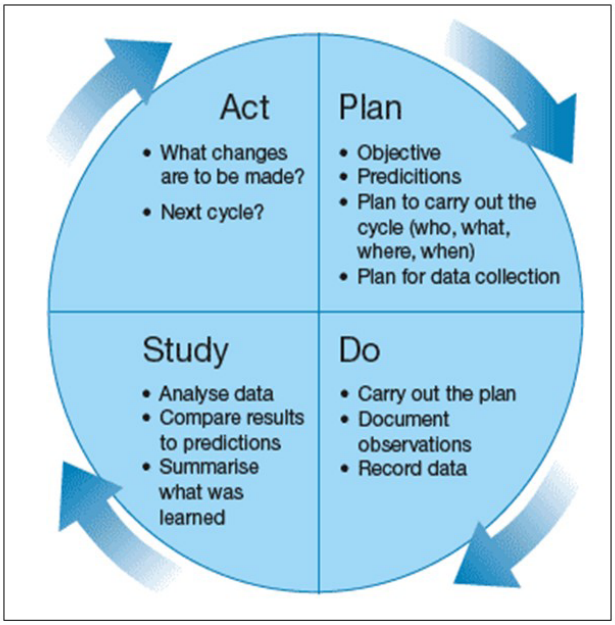
To assess pathway performance we retrospectively measured for three months prior to implementation of the change of practice and for three months following the implementation of the COVID-ADP the numbers of patients who had one or serial troponin tests within the ED, the numbers of patients admitted to hospital, the diagnoses of admitted patients (ICD10 codes), the numbers of patients discharged from ED, the lengths of stay of patients in the ED, the numbers of patients receiving next-day community troponin tests, and the repeat presentations to ED of all patients. The pathway was implemented on 6 May 2020. We compared the performance of the pathways from 6 February 2020 to 5 May 2020 (EDACS-ADP) and from 6 May 2020 to 6 August 2020 (COVID ADP). We excluded from the analysis all admitted patients with a myocardial infarction.

Our principal outcomes were length of stay, proportions of patients discharged from the ED, proportions of patients with single and serial troponin tests in the ED, proportions of patients admitted with an ultimate diagnosis of Unspecified or Other chest pain (ICD10 codes R07.3 and R07.4).

New Zealand has four COVID-19 alert levels (https://covid19.govt.nz/alert-system/about-the-alert-system/ - last accessed 23 Nov 2020).

In levels 3 and 4, people are instructed to stay at home in their bubble other than for essential personal travel. Healthcare services are to use virtual, non-contact consultations where possible.

Figure 1 **Change management process based on the Institute for Healthcare Improvement's PDSA Cycle and Kotter's 8 stages of change (11,12)**



Kotter's 8 Steps of Change

1. Create a Sense of Urgency
2. Build a Guiding Coalition
3. Form a Strategic Vision and Initiatives
4. Enlist a Volunteer Army
5. Enable Action by Removing Barriers
6. Generate Short-Term Wins
7. Sustain Acceleration
8. Institute Change

Visual illustration of A) PDSA cycle and B) Kotter's 8 Stages of Change

Supplementary Figure 1 **Stakeholders and stakeholder groups involved**

Emergency Department	Clinical staff, Clinical and Nursing Director, Pathway Developer, Nurse Educators, Research Nurses, phlebotomists, ward clerks	
	Dr Martin Than	Emergency Medicine Physician, Project Lead
	Dr Jacques Loubser	Emergency Medicine Physician, ED Co-lead
	Dr James Weaver	Clinical IT Lead
	Tracey Williams	Clinical Nurse Specialist, Cardiology portfolio
	Polly Grainger	ED Pathways and Forms Specialist
	Kay Ratahi	ED Supplies Specialist
Laboratory	A/Prof Chris Florkowski	Clinical Chemist, Canterbury Health Laboratories
	Vanessa Buchan	Operations Manager, Canterbury Health Laboratories
Community Laboratory	Liaison via Vanessa Buchan through Service Level Alliance	Southern Community Laboratories
Cardiology Department	Dr Sally Aldous	Cardiologist
	Dr John Lainchbury	Cardiologist
	Dr Phil Adamson	Cardiologist
	Dr Alison Nankivell	Advanced Trainee, Cardiology
	Dr Thomas Clendon	Advanced Trainee, Cardiology
	Margaret Cumming	Cardiology Charge Nurse Manager
	Rob Hallinan	Cardiology Service Manager
	Chest Pain Unit nursing staff	
Primary Care	Dr Andrew Meads	Clinical Director, Primary Care Acute Demand Service
	Debbie Krute	Nurse Leader, Primary Care Acute Demand Service
	Cathy Cooper	Nurse Leader, Primary Care Acute Demand Service
Research – Development of background dataset	Christchurch Heart Institute	
	Prof Chris Frampton	Biochemist
	Prof A Mark Richards	Cardiologist
	Prof Richard Troughton	Cardiologist
	Lorraine Skelton	Nurse Manager
	ED Science Group	
	Dr Joanna Young	Data Collection
	Antony Watson	Data Collection
	Felicity Turner	Data Collection
	Alieke Dierckx	Project Administrator
Data Science	Prof John Pickering	Acute Care Research Fellow
	Canterbury District Health Board Data Warehouse (Decision Support)	
	Scott Maxwell	Data Warehouse Manager
	Melanie Browne	Information Analyst
	Lesley Hamel	Project Specialist
	Soledad Labbe-Hubbard	Project Specialist
	Valentyna Sylevych	Performance Reporting Analyst
Management	Carolyn Gullery	General Manager Planning & Funding and Decision Support
	Dr Greg Hamilton	Change Transition Manager, Planning & Funding
	Harue Akimoto	Management Accountant
Exercise Stress Test / ECG Departments	ECG Technicians	
	Nursing staff	

At level four all businesses are closed except for essential businesses and all gatherings are cancelled and public venues closed.

All of New Zealand entered alert level 3 at 13:30 on 23 March 2020 and level 4 at midnight 25 March 2020. A state of emergency was declared on 24 March 2020. At midnight on 27 April New Zealand moved from alert level 4 to level 3 and from midnight of l3 May to level 2 and the state of emergency expired. Level 2 allows normal gatherings with <100 people.

All confidence intervals are 95% confidence intervals calculated using bootstrapping. The statistical calculations were made in R version 3.5 (10).

Change management process

The management of change was based on the principles set out by the well-established Plan-Do-Study-Act (PDSA) cycle model for improvement from the Institute for Healthcare Improvement (IHI), Figure 1 (11). It is a simple yet powerful tool for accelerating quality improvement. Once a team has set an aim, established its membership, and developed measures to determine whether a change leads to an improvement, then testing the change in the real work setting is possible.

The PDSA cycle is shorthand for testing a change—by planning it, trying it, observing the results, and acting on what is learned. We also used the principles based upon the 8-step process for leading change to plan the change management process (12). Many stakeholder relationships had already been established from previous 'chest-pain' pathway initiatives.

A revised stakeholder analysis was conducted (Supplementary Figure 1). After the decision logic for the new pathway was established and before full deployment occurred there was limited management of a number of cases by a small 'super-user' clinician group which helped to establish the practicality of the new approach and potential problems. For the first six weeks after deployment there were targeted stakeholder discussions, approximately weekly, to troubleshoot individual patient cases and/or clinician behaviours, after which a more ad hoc review process was adopted. All cases where a patient did not attend planned community follow-up troponin were reviewed by a Cardiology and/or Emergency Medicine Specialist and patient management decisions made according to their clinical judgement.

RESULTS

COVID-ADP development

There were 2416 subjects in the dataset used to develop the COVID-19 pathway of whom 38.2% were female and the mean age was 63 years (Table 1). Of these patients 452 (18.7%) had a MACE within 30d.

Amongst patients with EDACS < 16 and no new ischaemia on ECG there were 697 28.8% (95%CI: 27.1% to 30.6%) of all patients) patients with hs-cTnI <5 ng/L on first blood sampling (0h) with three (0.4% (0% to 1%)) 30d MACE. All were NSTEMI at initial hospital visit. The two-hour hs-cTnI concentrations for these three patients were 6, 17, and 225 ng/L all presenting ≥5h post-symptom onset. The overall strategy EDACS <16, no new ischaemia on ECG and 0h hs-cTnI <5 had a predicted sensitivity of 99.3% (95%CI: 98.1% to 99.8%) and Negative Predictive Value (NPV) or 99.6% (98.7% to 99.9%) (Aim 1).

Another 138 (5.7% (4.8% to 6.7%)) patients had a 0h hs-cTnI 5-14 ng/L with a three (2.2% (0 to 4.8%)) 30d MACE (Aim 2). The 30d MACE rate amongst the remaining 100 (4.1% (3.4% to 4.9%) of all patients) was 51% (41.3% to 61.0%). We determined patients in this group were to have an additional 2h troponin measurement and be assessed by Cardiology.

Table 1 Regulatory requirement of RECs

Demographic	Value
Age, years	63 +/- 13
Females	923 (38.2%)
Ethnicity	
New Zealand Māori	85 (3.5%)
Pacific	21 (0.9%)
New Zealand European	1745 (72.2%)
Other	268 (11.1%)
Unknown/Refuse to answer/Missing	297 (12.3%)
Diastolic Blood Pressure (mmHg)	81 +/- 14
Systolic Blood Pressure (mmHg)	147 +/- 26
Respiratory rate (breaths/minute)	17.2 +/- 3.5
O2 saturation (%)	97.3 +/- 1.9
Creatinine (umol/L)	93 +/- 30
Potassium (mmol/L)	4.1 +/- 0.5
White Cell Count (G/L)	7.8 +/- 2.6
Kilip class	
0	615 (25.6%)
I	1732 (72.0%)
II	55 (2.3%)
III	2 (0.1%)
Family history of Cardiovascular disease	1311 (54.3%)
History of Coronary artery disease	852 (35.3%)
History of Heart Failure	152 (6.3%)

History of Diabetes Mellitus	361 (15.0%)
History of Hypertension	1329 (55.0%)
History of Smoking	367 (15.2%)
History of Dyslipidaemia	1342 (55.6%)
Diaphoresis	1087 (45.0%)
Pain on Palpation	177 (7.3%)
Pleuritic pain	368 (15.2%)
Pain radiates to Arm, Neck or Jaw	1161 (48.1%)

Amongst patient with EDACS ≥16 or new-ischaemia on ECG there were 613 (25.4% of all patients) with 0h hs-cTnI <5 ng/L of whom 6 (1% (0.3% to 1.8%))) had a 30d MACE (5 index NSTEMI, 1 NSTEMI within subsequent 30d). The overall sensitivity for this sub-group was 98.7% (95%CI: 97.1% to 99.4%) and Negative Predictive Value (NPV) or 99.0% (97.9% to 99.6%). It was determined that these patients were to be discharged from ED with next-day follow-up troponin. Another 341 (21.1%) of patients had a 0h hs-cTnI 5-14 ng/L with a 8.2% (n=28) 30d MACE rate. This was considered too high for them to be all followed the next day, so they were further stratified by time from symptom onset <3h or >3h (Aim 3). In the ≤3h cohort there were 95 patients with a 15.8% (8.6% to 23.0%) 30d MACE rate. We determined patients in this group were to have an additional 2h troponin measurement and be assessed by Cardiology. There were 246 with time from symptom onset >3h with a 5.3% (2.7% to 8.2%) MACE rate. It was determined that these patients were to be discharged from ED with next-day follow-up troponin.

The 30d MACE rate among the remaining 528 (21.8% (20.2% to 23.5%)) patients (i.e. those with 0h hs-cTnI > 14 ng/L) was 68.4% (64.4% to 72.2%). It was determined that these patients were to be admitted to the Cardiology ward where they were to receive further troponin testing.

The change from using three EDACS strata (≤167, 16-20, ≥21; Figure 2) to two EDACS strata (≤16, >16; Figure 2) reduce the need for both cardiology and emergency department teams to assess the same patients (compare Figures 2 and 3) (Aim 5). This resulted in predicted change in duplicate clinical assessment from 20% to 4%. In our development dataset 935 (38.7%) had EDACS <16, therefore in the COVID-ADP would be expected to be evaluated by ED physicians, whereas (61.3%) would be expected to be evaluated by Cardiology.

Overall 28.8% of patients could be discharged from the ED without follow-up on the basis of a single hs-cTnI with a 30d MACE rate of 0.4%. A further 41.2% could be discharged from the ED with next-day follow-up with a 4.9% 30d MACE rate. The final pathway is shown in figure 3. This meant that ~70% of patients would not require measurement of a second troponin measurement while in hospital.

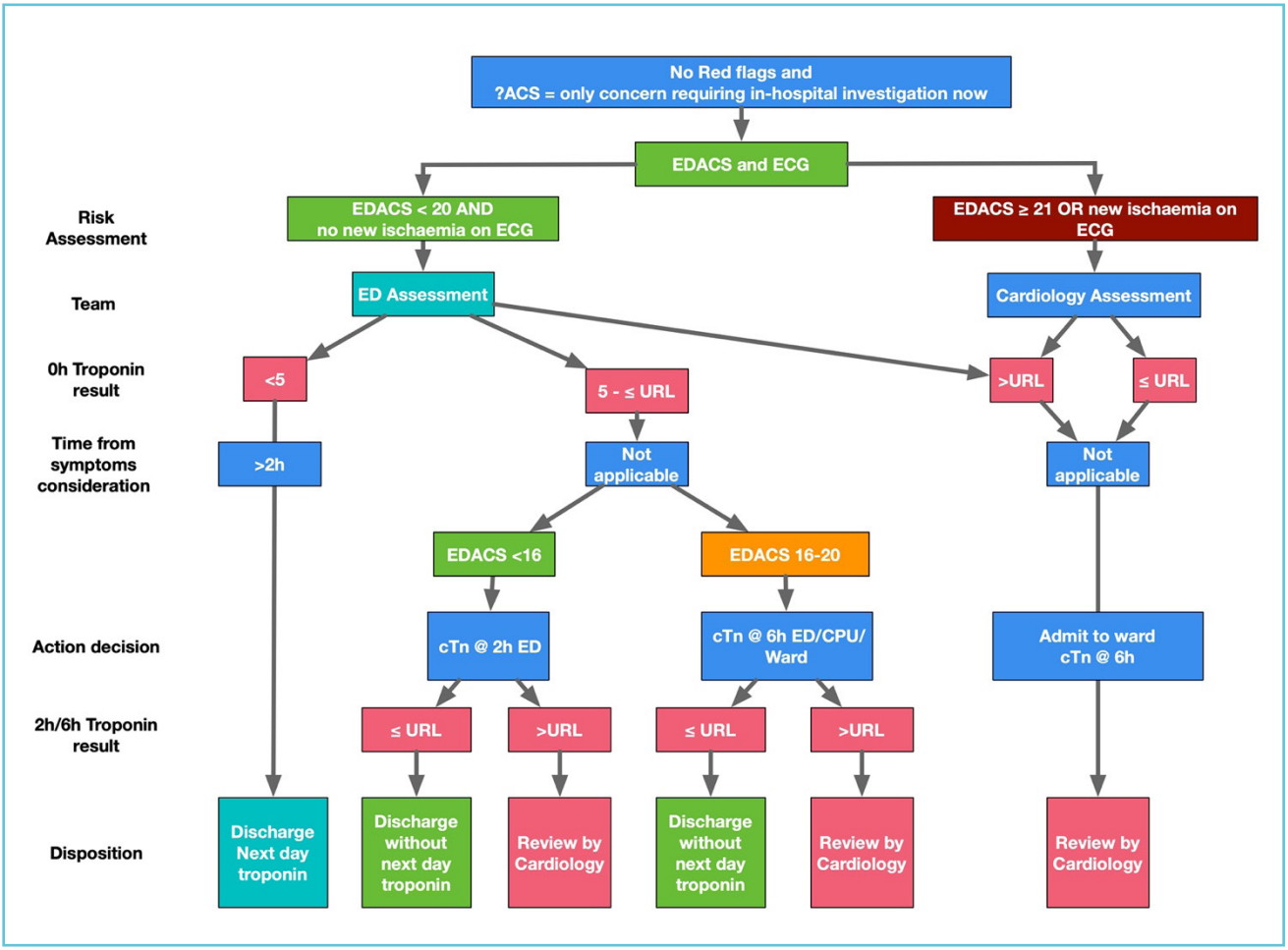
The EDACS-ADP for patients having a next-day community test used the sex-specific URL or significant rise as a trigger for cardiology review. For the COVID-ADP we used the results of patients

who had had a diagnosis of a MACE and serial troponin measurements with the first being 5-14 ng/L to determine a change in troponin concentration of ≥ 4 ng/L at which to trigger a review by Cardiology (Aim 4).

COVID-ADP implementation and performance

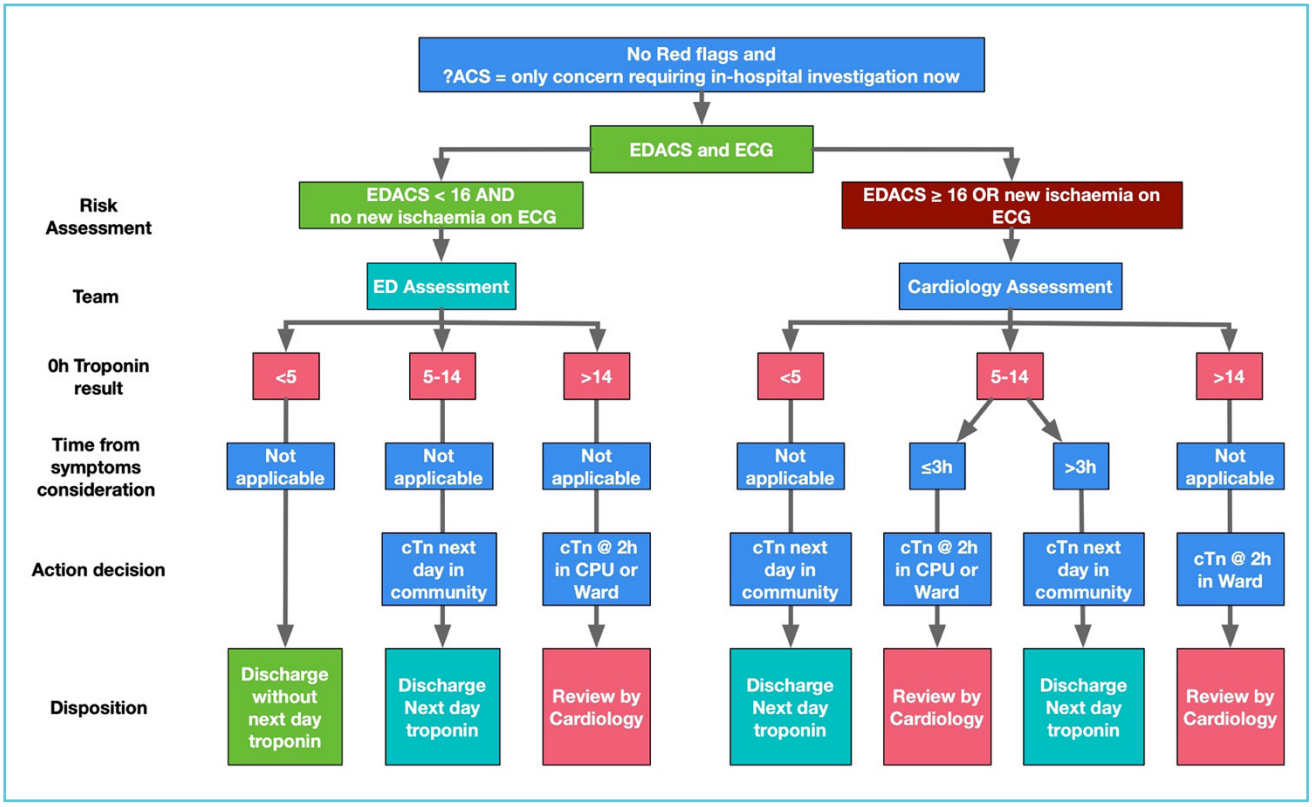
In the three months prior to COVID-ADP implementation 1,073 people presented with a primary complaint of Chest Pain compared with 1,343 post COVID-ADP implementation. The primary reason for the 25.2% increase post-implementation is that, during the period from 23 March to 13 May, New Zealand was under heavy COVID-19 lock-down restrictions. There could also be a seasonal effect. Despite this increase in Chest Pain presentations post-implementation there was an 8% reduction in the numbers of patients admitted who were admitted and ultimate diagnosed with Unspecified or Other chest pain. We accounted for the post-implementation increase in Chest Pain presentations by using the rate of Chest Pain presentations as a denominator. Consequently, the rate of Unspecified or Other chest pain admissions to Chest Pain presentations decreased from

Figure 2 **The EDACS-ADP. The 2h cTn is 2h after the first blood draw for the 0h cTn. The 6h cTn is at least 6h after symptom onset or worst symptom if later**



ACS: Acute Coronary Syndrome. cTn: cardiac Troponin. URL: Upper Reference Limit (Sex specific for hs-cTnI; 16ng/L for females, 34ng/L for males). ECG: Electrocardiogram. EDACS: Emergency Department Assessment of Chest pain Score. CPU: Chest Pain Unit.

Figure 3 The COVID-ADP



ECG: Electrocardiogram. EDACS: Emergency Department Assessment of Chest pain Score.

12.3% to 9.3% representing an estimated 24.3% reduction in admissions with Unspecified or Other chest pain (non-cardiac).

In the target group of Chest Pain presenters discharged from the ED there was an immediate increase in the number with only a single troponin test in the ED, Figure 4. In the 3 months prior to the implementation of the COVID-ADP (EDACS-ADP era) 579 had a single troponin in the ED, in the 3 months with the COVID-ADP 954 had a single troponin representing a 64.7% (~1.6 fold) increase. To account for the reduced presentations during lockdown we looked at the proportion of patients with two troponin tests in the ED because a decrease in this proportion represents a decrease in the number of patients requiring further evaluation beyond the first blood test. Figure 5 illustrates that there was an immediate dramatic reduction, from a weekly mean of 29.6% to a weekly mean of 11.7%, representing a 60% reduction in patients requiring 'two-troponin' evaluation for possible ACS.

The median (lower-quartile – upper-quartile) length of ED stay (LOS) of all patients with a troponin test in the ED reduced from 3.8h (2.8h – 4.9h) to 3.4h (2.6h – 4.6h). A Mann-Whitney test of length of stay (LOS) demonstrated that the before and after implementation distribution of lengths of stay are not the same ($p<0.0001$). In the target group of those discharged from ED the median LOS prior to implementation was 3.7h (2.7h - 4.6h) compared to the post implementation LOS of 3.1h (2.4h – 4.1h). The mean LOS was 3.9h for the current pathway and 3.4h for the COVID-ADP suggesting an average time saving of 30 minutes per patient. The proportion of the target group patients discharged from ED within 2h increased post-implementation from

5.6% to 8.1%, a 44.6% increase. The proportion discharged from ED within 3h increased post-implementation from 32.7% to 44.2%, a 35.2% increase.

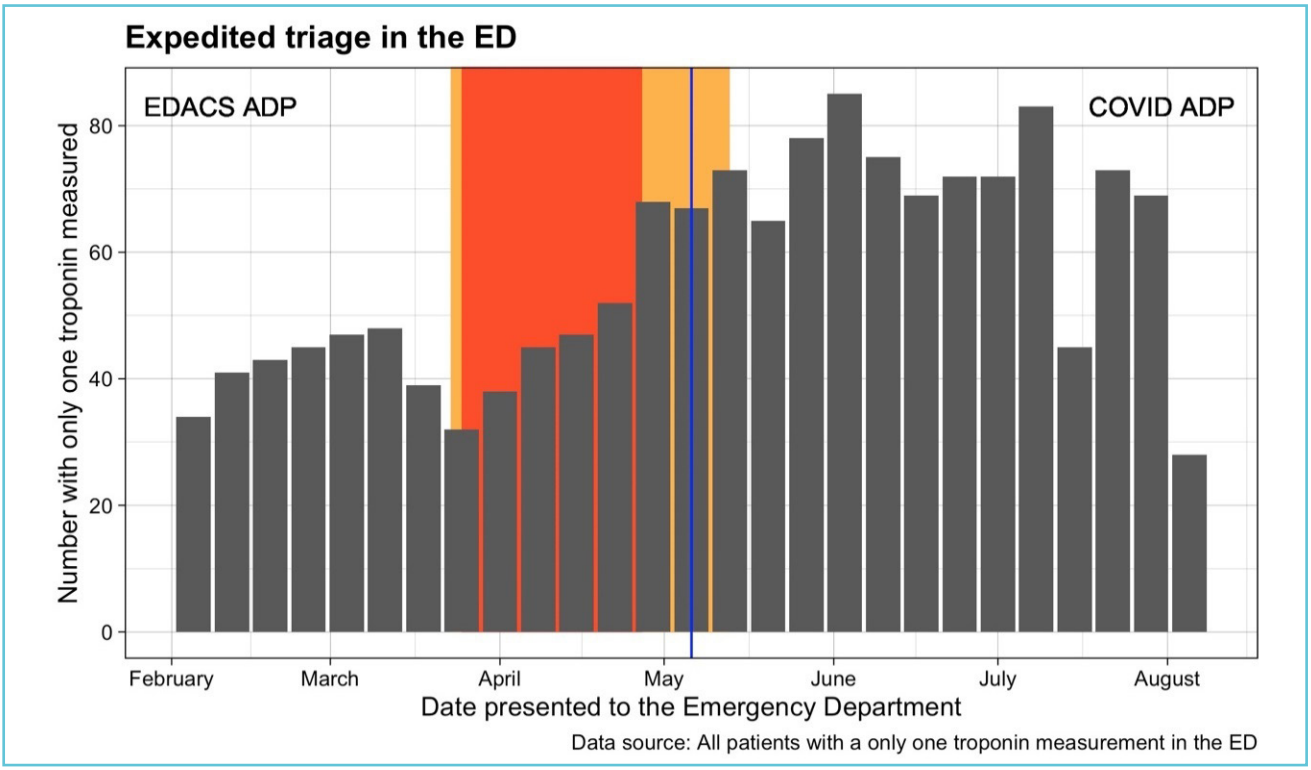
There were two patients who had an MI within 7 days of first presentation who had been discharged from the ED. One of these had self-discharged from the ED against medical advice. The other patient's initial hs-cTnI was less than the LoD. While this presentation was after implementation of the COVID-ADP they were equally eligible for early discharge under the former pathway. They presented 5 days later with more chest pain and an initial troponin concentration of 15 ng/L which rose to 21 ng/L.

In 2019 there were 10,548 presentations to the ED with at least one troponin measurement. Of these 1,249 (11.8%) were admitted to hospital and ultimately discharged after a mean 28h stay with a diagnosis of Unspecified or Other chest pain. If we apply our finding of a 24% reduction in admission of this patient group this would mean an annual 300 fewer patients spending a day in hospital. This would be a cost saving of NZ$390,000. The 30-minute average reduction of time spent in ED translates to 4852 fewer hours of patient time in the ED per annum and an estimated saving of NZ$146,000 bringing the total annual saving to the health system to over NZ$0.5Million.

DISCUSSION

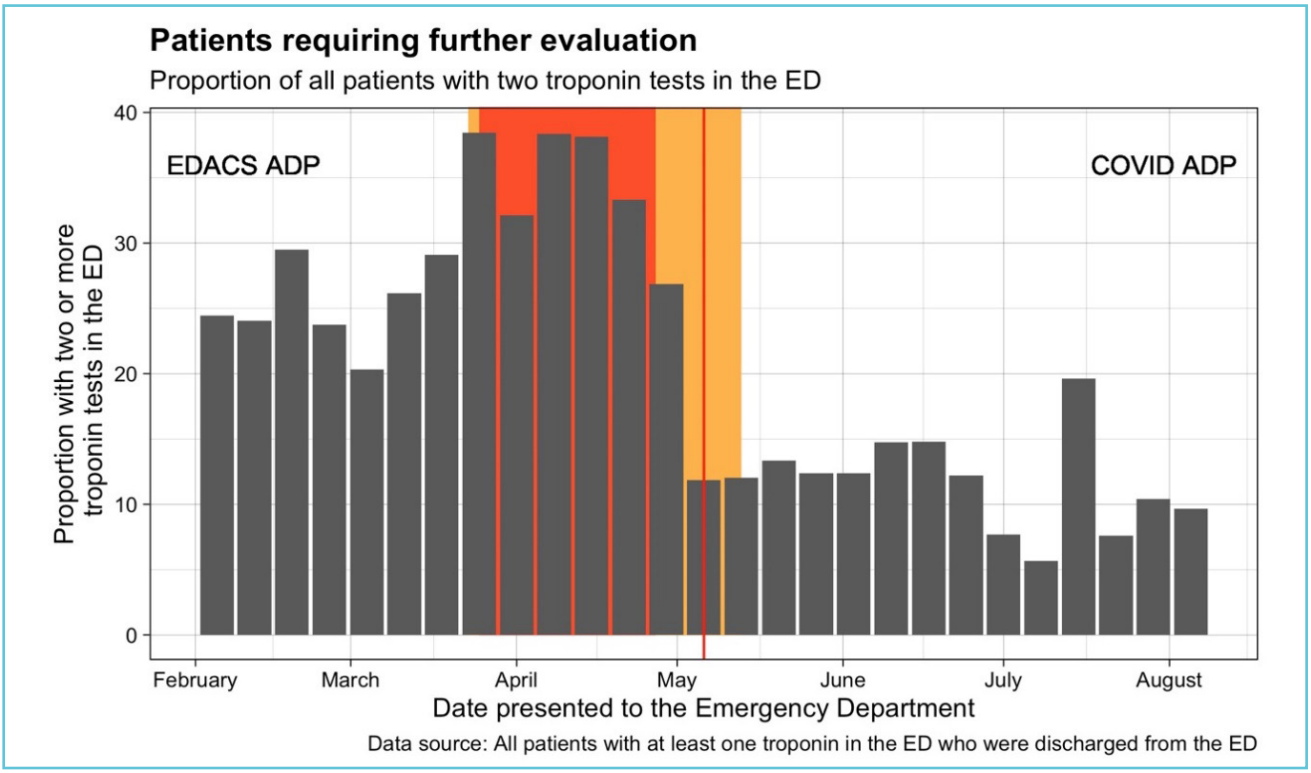
We have demonstrated that it was possible, in real life care, to make rapid changes to an accelerated diagnostic pathway which made an immediate and meaningful impact to patient care. We

Figure 4 More presenters had only one troponin measurement with the COVID-ADP compared to the EDACS ADP



Blue line: 6 May, date of implementation of COVID-ADP.
Orange: New Zealand pandemic alert level 3; Red: Alert level 4.

Figure 5 **Fewer of the presenters who were discharged home from the ED needed evaluation with a second troponin measurement with the COVID-ADP compared to the EDACS-ADP**



Blue line: 6 May, date of implementation of COVID-ADP.
Orange: New Zealand pandemic alert level 3; Red: Alert level 4.

were able to markedly reduce the use of repeat troponin testing in hospital which led to reduced time in ED and reduced admissions for patients without cardiac chest pain. This was important because we were able to reduce the potential risk of cross-infectivity and hospital resource burden for this common patient group.

There are a number of key take-aways from this project that inform rapid change management in general. *Firstly*, the objectives of the project (reduced length of stay and admission rates) were clearly established from the beginning by consensus. *Secondly*, it was agreed that reduction of serial testing would be the optimal way to achieve these objectives. *Thirdly*, we had access to high quality data enabling us to predict downstream event rates for the patients and so establish an evidence base for proposed changes. *Fourthly*, we used wide cross-stakeholder consensus to agree specified actions for patients within different risk strata and then worked backwards using the data to determine how to identify which patients fitted within each risk group. *Fifthly*, we placed a high level of importance upon effective change management processes. *Sixthly*, the time invested in the preceding years to build meaningful and agile stakeholder relationships made possible the expedited change management processes in response to the pandemic situation.

Finally, video conferencing enabled robust discussion amongst multiple stakeholders when face to face meetings were not possible. Notably, by meeting remotely, it was possible to have a much more frequent meeting schedule than had

been previously possible when requiring coordination of schedule for face-to-face meetings. This, in itself, allowed an acceleration of previous decision-making and change-management processes.

This project has a number of strengths. Most importantly perhaps, our project describes what actually happened to patient care. We evaluated this project using data collected prospectively and routinely as part of healthcare delivery. Because we were able to base this change on extant well researched data and have shown it to be safe and effective, we believe that this project and its onward monitoring is both sustainable and transferable to other centres. Additionally, although not an original objective, the project reduced costs for the healthcare system.

There are some limitations. Firstly, the use of routinely collected data to evaluate the change required us to infer the group being assessed for possible myocardial infarction based on the measurement of troponin. Therefore, we may be overestimating the numbers of patients actually investigated for possible myocardial infarction. The same inference was used for both the time period before and after the COVID-ADP implementation, thus any overestimate will not affect our conclusions of a successful change of practice. Secondly, while the data sets used to derive the pathway have been well described in multiple publications in the literature and have only limited exclusions, they are nevertheless not exactly representative of all patients being evaluated for possible myocardial infarction.

CONCLUSION

Strong stakeholder relationships and change management processes, video conferencing and access to high quality data allowed rapid and agile re-design and implementation of a chest pain assessment pathway without face-to-face contact. Significant meaningful impact was demonstrated resulting in the pathway being permanently adopted despite the relaxation of the easing of the alert levels and cessation of the need for an immediate response to the pandemic.

❖❖❖❖❖

Acknowledgments and disclosures

Many thanks to all multidisciplinary team members associated with this project, many of which are listed below who have each collectively helped us advance care and achieve a healthcare excellence award as one of the top three winners of the UNIVANTS of Healthcare Excellence™ awards in 2020 (13). Thanks to the following: Melanie Browne, Vanessa Buchan, Cathy Cooper, Margaret Cumming, Alieke Dierckx, Carolyn Gullery, Lesley Hamel, Dr. Greg Hamilton, Debbie Krute, Soledad Labbe-Hubbard, Sandra English, Scott Maxwell, Dr. Andrew Meads, Prof. Chris Pemberton, Kay Ratahi, Prof. Mark Richards, Valentyna Sylevych, Prof. Richard Troughton, Felicity Turner, Antony Watson, Dr. James Weaver, Tracey Williams.

❖❖❖❖❖

REFERENCES

1. Goodacre S, Cross E, Arnold J, Angelini K, Capewell S, Nicholl J. The health care burden of acute chest pain. Heart. 2005;91:229–30.

2. Pitts SR, Niska W, Burt CW. National Hospital Ambulatory Medical Care Survey: 2006 Emergency Department Summary. National Health Statistics Report. 2008;7:1–39.

3. Than MP, Aldous S, Lord SJ, Goodacre S, Frampton CMA, Troughton R, et al. A 2-hour diagnostic protocol for possible cardiac chest pain in the emergency department: a randomized clinical trial. JAMA Intern Med. 2014;174:51–8.

4. Than MP, Pickering JW, Aldous SJ, Cullen LA, Frampton CMA, Peacock WF, et al. Effectiveness of EDACS versus ADAPT accelerated diagnostic pathways for chest pain: A pragmatic randomized controlled trial embedded within practice. Annals of Emergency Medicine. 2016;68:93-102.e1.

5. Young JM, Sage JN, Pickering JW, Bannister L, Aldous S, George PM, et al. Next day troponin tests in real world implementation of baseline troponin rule-out of myocardial infarction demonstrates minimal delayed troponin rises. Circulation. 2020;In Press.

6. Chapman AR, Lee KK, McAllister DA, Cullen L, Greenslade JH, Parsonage W, et al. Association of high-sensitivity cardiac troponin I concentration with cardiac outcomes in patients with suspected Acute coronary syndrome. JAMA. 2017;318:1913.

7. Neumann JT, Twerenbold R, Ojeda F, Sörensen NA, Chapman AR, Shah ASV, et al. Application of High-Sensitivity Troponin in Suspected Myocardial Infarction. N Engl J Med. 2019;380:2529–40.

8. Than MP, Cullen LA, Aldous S, Parsonage WA, Reid CM, Greenslade JH, et al. 2-Hour accelerated diagnostic protocol to assess patients with chest pain symptoms using contemporary troponins as the only biomarker: the ADAPT trial. JACC. 2012;59:2091–8.

9. Pickering JW, Young JM, George PM, Pemberton CJ, Watson A, Aldous SJ, et al. Early kinetic profiles of troponin I and T measured by high-sensitivity assays in patients with myocardial infarction. Clinica Chimica Acta. 2020; S0009898120300644.

10. R Core Team. R: A language and environment for statistical computing [Internet]. Vienna, Austria: R Foundation for Statistical Computing; Available from: http://www.R-project.org/

11. Institute for Healthcare Improvement: PDSA Cycle [Internet]. www.ihi.org. [cited 2020 Jan 27]. Available from: http://www.ihi.org/resources/Pages/HowtoImprove/default.aspx

12. Kotter JP. Leading Change. Boston: Harvard Business School Press; 1996.

13. Strain C and Ravalico TH. Laboratory medicine and healthcare excellence; Till death do us part. eJIFCC 2021; 32:1:007-019.