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Implementation and evaluation of a rural general practice assessment pathway for possible cardiac chest pain using point-of-care troponin testing: a pilot study

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ABSTRACT

Objectives To assess the feasibility and acceptability, and additionally to preliminarily evaluate, the effectiveness and safety of an accelerated diagnostic chest pain pathway in rural general practice using point-of-care troponin to identify patients at low risk of acute myocardial infarction, avoiding unnecessary patient transfer to hospital and enabling early discharge home.

Design A prospective observational pilot evaluation.

Setting Twelve rural general (family) practices in the Midlands region of New Zealand.

Participants Patients aged ≥18 years who presented acutely to rural general practice with suspected ischaemic chest pain for whom the doctor intended transfer to hospital for serial troponin measurement.

Outcome measures The proportion of patients managed using the low-risk pathway without transfer to hospital and without 30-day major adverse cardiac event (MACE); pathway adherence; rate of 30-day MACE; patient satisfaction with care; and agreement between point-of-care and laboratory measured troponin concentrations.

Results A total of 180 patients were assessed by the pathway. The pathway classified 111 patients (61.7%) as low-risk and all were managed in rural general practice with no 30-day MACE (0%, 95% CI 0.0% to 3.3%). Adherence to the low-risk pathway was 95.5% (106 out of 111). Of the 56 patients classified as non-low-risk and referred to hospital, 9 (16.1%) had a 30-day MACE. A further 13 non-low-risk patients were not transferred to hospital, with no events. The sensitivity of the pathway for 30-day MACE was 100.0% (95% CI 70.1% to 100%). Of low-risk patients, 94% reported good to excellent satisfaction with care. Good concordance was observed between point-of-care and duplicate laboratory measured troponin concentrations.

Conclusions The use of an accelerated diagnostic chest pain pathway incorporating point-of-care troponin in a rural general practice setting was feasible and acceptable, with preliminary results suggesting that it may safely and effectively reduce the urgent transfer of low-risk patients to hospital.

Strengths and limitations of this study

- This is the first 'real-life' pilot evaluation of a clinical accelerated diagnostic chest pain pathway incorporating point-of-care troponin testing that was specifically created for rural use in the general practice setting.
- This pilot implementation allows assessment of the feasibility and acceptability of a structured clinical pathway for chest pain assessment designed to reduce unnecessary transfer to hospital and allow patient management in rural general practice, and included monitoring of clinician compliance and patient satisfaction.
- The small sample size limits the generalisability of this pilot evaluation but informs larger studies to be undertaken to confirm the safety and efficacy of this chest pain pathway in rural settings.

INTRODUCTION

Chest pain with symptoms suggestive of acute coronary syndrome (ACS) is one of the most common reasons for presentation to emergency departments (EDs) in New Zealand (NZ) and across the developed world. More than 80% of such cases, however, do not have a final diagnosis of acute myocardial infarction (AMI).[1–5] The AMI rate is even lower in patients presenting to general practice with chest pain.[1 6–8] Assessment of such patients represents a major clinical challenge for clinicians as a missed diagnosis can lead to adverse cardiac outcomes, including death.[4 9–11] Historically, this has led to prolonged investigation and length of stay, contributing to overcrowding in EDs, a high resource burden on health systems and adverse patient outcomes.[4 10–14]

NZ has been an international leader in developing and implementing validated accelerated diagnostic pathways (ADPs) for the hospital assessment of patients presenting with suspected cardiac ischaemia.[15–17] These ADPs combine structured scoring of clinical variables, electrocardiographic interpretation and cardiac troponin testing to identify patients at low risk of AMI.[15–17] All metropolitan EDs across NZ have adopted ADPs, as recommended by national health policy. This followed randomised controlled trials and successful implementation of ADPs at Christchurch Hospital (Christchurch, NZ) which demonstrated improvement in early safe discharge rates from less than 10% to over 30% of patients.[15 16] In metropolitan NZ hospitals, implementation of these clinical pathways doubled the proportion of patients with chest pain discharged from EDs within 6 hours, without affecting the 30-day major adverse cardiac event (MACE) rates.[17]

ADPs used in NZ require immediate access to laboratory-based contemporary or high-sensitivity troponin assays.[17] However, the majority of rural-based general practitioners do not have timely access to these laboratory assays.[18] Point-of-care cardiac troponin (POC-cTn) assays are less precise and have lower analytical sensitivity than laboratory-based assays and therefore have not been considered appropriate for assessment of possible ACS in general practice.[19]

The lack of evidence-based pathways for POC-cTn assays used in rural general practice and the geographical challenges of rural NZ result in often long (up to 3 hours) and difficult road transfer to hospital or risk potentially missed AMI. This has an impact on acute healthcare demand in the Midland Health region (Bay of Plenty, Taranaki, Tairāwhiti and Waikato District Health Boards), which includes a significant number of rural communities. Similar issues have been described for access to prehospital and advanced level emergency care in NZ.[20]

There is evidence in urban EDs that the use of POC-cTn assays for identification of low-risk patients, as part of an ADP that includes a clinical score, paired POC-cTn assays 2 hours apart and ECGs, is safe.[21 22] Australasian guidelines support reducing the cut-off below the manufacturer's recommendation to improve clinical sensitivity.[19]

Adoption of ADPs for suspected cardiac chest pain by NZ rural general practices has the potential to reduce health service burden and improve patient outcomes by avoiding unnecessary transfers and admissions to regional or base hospitals, providing early reassurance to low-risk patients who can be safely managed closer to home in primary care, while expediting treatment of patients at non-low-risk of AMI.

Aims

- To pilot the real-life feasibility and acceptability of implementing an ADP using POC-cTn to identify low-risk patients in rural general practice who do not require urgent transfer to hospital for further cardiac work-up.
- To make a preliminary assessment of the effectiveness and safety of implementing a structured chest pain pathway in rural general practice.

METHODS

Study design

This is a prospective pilot evaluation of implementing as standard care an ADP using POC-cTn specifically adapted for management of suspected cardiac chest pain in rural general practice, from 31 October 2016 with data collection/monitoring continued to 31 July 2018. The pathway was modified from a validated metropolitan ED chest pain ADP[16] and incorporated the Emergency Department Assessment of Chest Pain Score (EDACS) (online supplemental table 1), ECG and POC-cTn measurements at presentation and 2 hours at the practice (figure 1). The EDACS-ADP was developed with contemporary troponin assays and has been validated for use with both contemporary and high-sensitivity assays.[2 15 16 23]

Setting and location

The pilot evaluation was undertaken in 12 rural general practices from the Pinnacle Midlands Health Network, a primary healthcare organisation in NZ (online supplemental table 2). There is one metropolitan ED (Waikato Hospital) and four rural hospitals (Te Kuiti, Tokoroa, Thames, Taumarunui) to which patients may be transferred for assessment and treatment. The participating practices had expressed an interest or had been identified as having high volumes of presentations and did not have have access to testing with POC-cTn prior to pathway implementation. They had a diverse demographic and population size (range 2670–10 000 patients). The ADP was implemented in weekly tranches of three practices from 31 October 2016 to 21 November 2016 (online supplemental table 3). One practice withdrew due to an inability to enrol subjects within the required period. The evaluation period was for 22 months, with follow-up completed by 30 August 2018.

On-site training and performance of i-STAT testing were provided by an Abbott Point of Care representative to the site staff identified as primary users prior to implementation of the chest pain pathway. The training also consisted of 'train the trainer', where key superusers at each site were trained to be able to carry out training to any staff who missed out of the original training. The superusers were also trained to run the control material for quality control new cartridge stock. The i-STAT was only used to measure troponin concentrations.

Patients

All patients identified and managed according to the pathway were provided with written information about the new model of care for suspected cardiac chest pain and follow-up.

Inclusion criteria

The inclusion criteria were patients aged ≥18 years who presented acutely to rural general practice with suspected

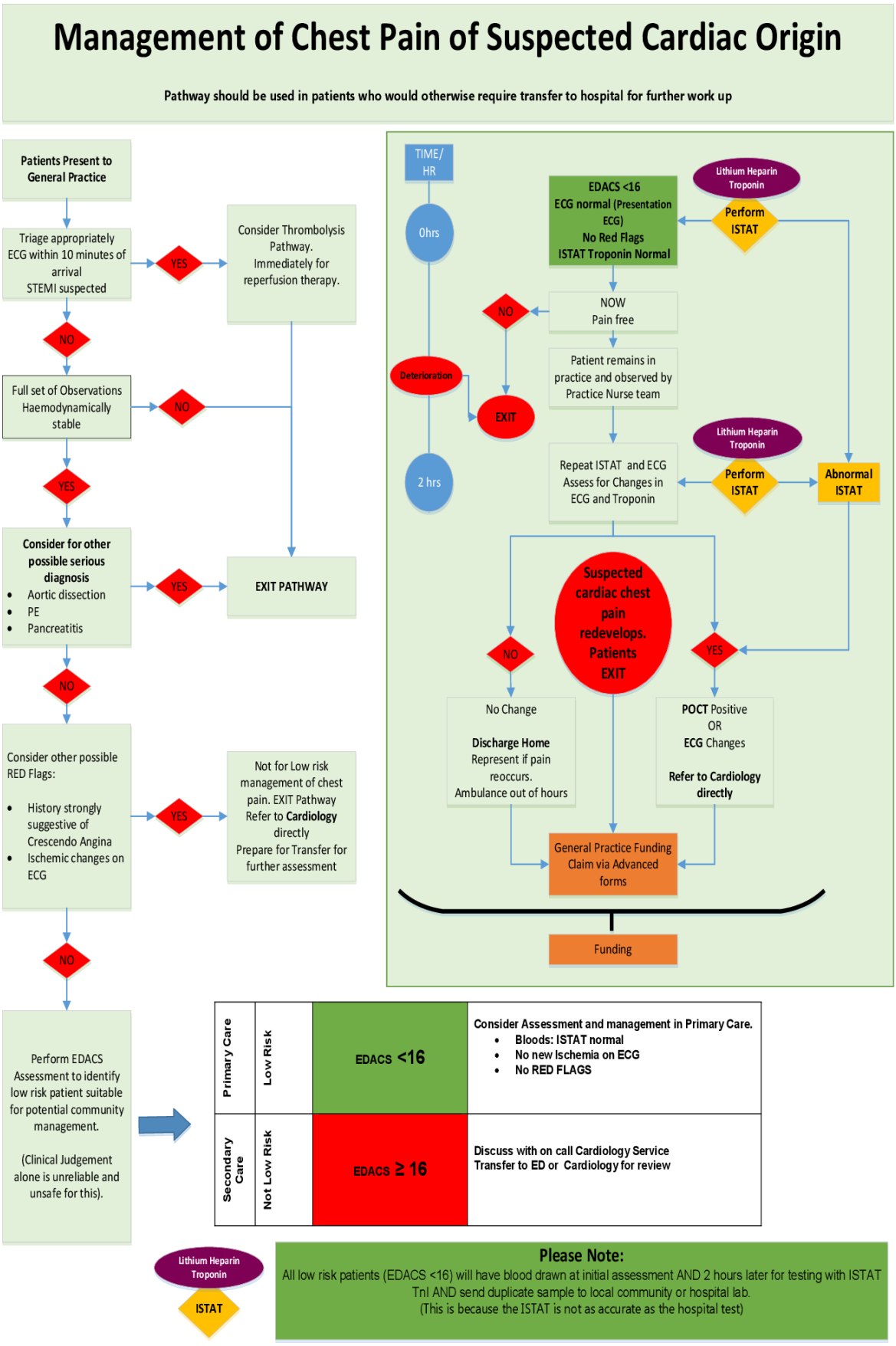

Figure 1 Flow diagram of recommended clinical management according to risk categorisation for the rural accelerated diagnostic chest pain pathway. ED, emergency department; EDACS, Emergency Department Assessment of Chest Pain Score; STEMI, ST-segment elevation myocardial infarction.

cardiac ischaemia (primarily chest pain) who the doctor would ordinarily transfer to hospital for serial troponin measurement.[24]

Exclusion criteria

Patients with any of the following were excluded: (1) ST-segment elevation myocardial infarction; (2) proven or suspected non-coronary pathology as the cause of chest pain (eg, pancreatitis or pulmonary embolism); (3) requiring transfer to hospital regardless of a POC-cTn below the prespecified threshold, due to other medical conditions, or the need for other investigations or were systemically unwell; (4) chest pain symptoms greater than 72 hours; (5) representation with chest pain symptoms during the evaluation period; or (6) an anticipated problem with follow-up (eg, resident outside NZ).

Patient and public involvement

Patient satisfaction and acceptability of the new model of care were addressed by phone questionnaire at 30-day follow-up for those who underwent assessment via the pathway at the participating practices. It was not appropriate or possible to involve patients or the public in the design, or conduct, or reporting or dissemination plans of our research.

Rural accelerated diagnostic chest pain pathway

A programme of face-to-face education sessions regarding the pathway was provided to the staff at the participating practices.

Identification and management of low-risk patients

Patients had to meet all the following features to be categorised as low-risk:

- No red flags (history of ongoing pain or haemodynamically unstable or history suggestive of crescendo angina).
- Absence of possible new ischaemic changes on ECG: ST-segment depression (≥0.5 mm) in two contiguous leads (including reciprocal changes), abnormal T-wave inversion (≥2 mm), Q-waves (>30 ms and ≥0.1 mV depth) in two contiguous leads, or new bundle-branch block on an ECG at 0 and 2 hours.
- EDACS <16 (online supplemental table 1).[23 25]
- POC-cTn: Abbott i-STAT cTnI <40 ng/L (decision limit) at 0 and 2 hours.
- No further chest pain or ECG changes and remained clinically stable.

Patients meeting the above low-risk criteria were eligible for discharge from the rural general practice to home, with follow-up outpatient exercise tolerance testing where indicated.

Identification and management of non-low-risk patients

Patients who did not fulfil the criteria for the low-risk pathway were referred to hospital for assessment and serial troponin testing.

POC-cTnI testing

Venous blood sampling was undertaken for point-of-care cardiac troponin I (POC-cTnI) testing at presentation and after 2 hours of presentation to each participating rural general practice and analysed on an Abbott i-STAT. The Abbott i-STAT c-TnI assay has a limit of detection (LoD) of 20 ng/L, limit of quantitation of 40 ng/L and 99th percentile at 80 ng/L.[25] The Australian Academy of Clinical Biochemistry has recommended 40 ng/L as a suitable decision limit for assessment of AMI,[18] which was adopted for this pathway. The i-Stat analysers were installed by an Abbott representative, who also provided and certified competency for the users, who performed daily electronic quality control (QC) and tested liquid QC samples on receipt of each batch of cartridges and on a monthly basis.

Duplicate samples at both timepoints were sent to either the local hospital laboratory for Roche Diagnostics Elecsys high-sensitivity cardiac troponin T (hs-cTnT), with an LoD of 5 ng/L and 99th percentile at 14 ng/L,[25] or the local community laboratory for Beckman Coulter Access cardiac troponin I (cTnI), with an LoD of 2.5 ng/L and overall 99th percentile at 17.5 ng/L[25] testing, as a safety audit measure. The Roche hs-cTnT assay thresholds were ≤14 ng/L for both genders and the Beckman cTnI assay thresholds were <10 ng/L for women and <20 ng/L for men. The results of point of care troponin (POCT) tests and the central laboratory analysis of duplicate samples were compared on a daily basis. Patients identified as low-risk and discharged directly home from general practice but with a subsequently elevated laboratory troponin result were contacted and referred for urgent hospital review.

Outcome measures

Implementation outcomes

- Adherence to the pathway.
- Patient acceptability and satisfaction with care.
- Participating sites' acceptability.

Intervention outcomes

- The proportion of patients identified as low-risk by the pathway, and managed in the community, without transfer to hospital, with no 30-day MACE.
  MACE was defined as death that was not known to be from non-cardiac causes, emergency coronary revascularisation procedure, cardiac arrest (International Classification of Diseases, 10th Version codes I460, I461, I469), and AMI[26 27] (I210, I211, I212, I213, I214, I219, I220, I221, I228, I229), ventricular arrhythmia (I472), cardiogenic shock (R570) and high-degree atrioventricular block needing intervention (I4442).
- MACE within 30 days of presentation in non-low-risk patients.
- ACS (AMI or unstable angina) within 30 days of presentation in non-low-risk patients.
- Non-emergency coronary revascularisation within 30 days of presentation in non-low-risk patients.

- Agreement between POC and laboratory measured cardiac troponin concentrations.

Data collection

Data were captured via a customised electronic template built into the practice management system (Medtech32) that contained embedded guidance for clinical decision making. Patient data were uploaded from the practice management system template into a secure web-based notebook hosted by the Pinnacle Midlands Health Network (a primary healthcare organisation). A research nurse performed telephone follow-up at 30 days to complete the patient satisfaction survey and identify any patient-reported cardiac events, hospital presentations or procedures that occurred following the general practice index presentation. The presence of 30-day MACE was collected from the hospital information systems using National Health Index (NHI) identifier event searches. NZ's NHI enables the identification of all hospital admissions or deaths throughout the country. Distances and time of transfer of patients from each general practice to the relevant base or rural hospital were derived from Google Maps. Data from all sources were combined in a secure database and analysed. De-identified data will be stored securely for a period of 10 years.

Sample size

A sample size of 200 patients was estimated to provide at least 70 low-risk patients by the rural accelerated chest pain pathway. Thus, assuming 35% patients could be managed in the community,[23] a sample size of 200 patients would generate a 95% CI of the estimate of approximately ±5% for the percentage of low-risk patients.

Statistical analyses

The proportion of low-risk patients identified by the ADP and managed in the rural general practice setting without transfer to hospital or occurrence of MACE within 30 days was estimated with a 95% CI. Sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) were derived. Other outcomes were summarised as means, medians and percentages, as appropriate with 95% CIs. Comparison of the proportion of variables between different groups was tested with independent t-test, Fisher's exact test or Mann-Whitney U test, as appropriate.

RESULTS

Study patients

There were 186 patients identified using the clinical pathway. Of these, six were excluded due to possible ST-elevation MI (n=2), alternative serious differential diagnosis (n=2), insufficient time for protocol completion at the practice (n=1) and chest pain representation (n=1). Therefore, a total of 180 patients were included in the analysis and 30-day follow-up was completed for all patients. Of the patients, 111 (61.7%) were classified as low-risk by the pathway and 69 (38.3%) as non-low-risk (figure 2).

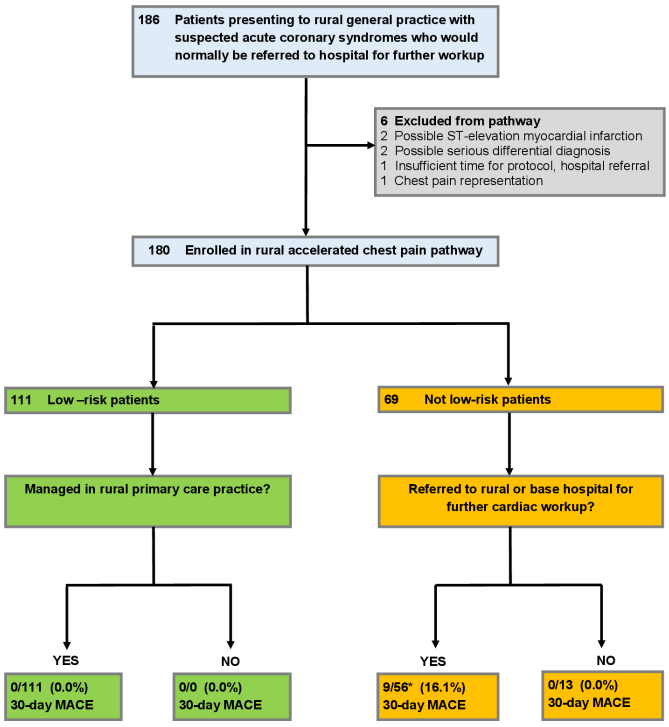

Figure 2 Flow of patients presenting to 11 participating rural general practice care practices with suspected chest pain through the rural accelerated chest pain pathway in the Midlands region of New Zealand. No patients were lost to follow-up. *One patient had a diagnosis of NSTEMI at index presentation and a second NSTEMI during 30-day follow-up. MACE, major adverse cardiac event; NSTEMI, non-ST-elevation myocardial infarction.

Non-low-risk patients were older, more likely to be male, and more likely to have cardiovascular risk factors or a history of ischaemic heart disease than low-risk patients (table 1). The median time from index chest pain onset to presentation to rural practice was 15 hours (IQR: 3.1–40.2 hours). Forty-two patients (23.3%) presented within the first 3 hours from symptom onset, of whom 22 were classified as non-low-risk.

Outcomes

Implementation outcomes

Adherence to pathway

Of the 111 low-risk patients, 106 (95.5%) adhered to the pathway, with 5 patients not completing the 2-hour assessment due to a subsequent diagnosis of non-cardiac chest pain (n=4) or refusal to remain in the practice (n=1). There were 13 (18.8%) patients classified as non-low-risk who were not transferred for hospital assessment, against pathway guidance. Of these, nine were classified as non-low-risk based on EDACS ≥16 alone; one patient had possible new ischaemic changes on ECG; three had ongoing chest pain, haemodynamic instability or history of crescendo angina; and one had an EDACS ≥16, together with ongoing chest pain and haemodynamic instability. None of these patients had elevated troponin

Table 1 Presenting features of patients

Characteristics	Low-risk (n=111)	Non-low-risk (n=69)	Difference between low-risk vs non-low-risk, % for counts and absolute difference for age (95% CI)	Non-low-risk, referred to hospital (n=56)	Non-low-risk, managed at home (n=13)
Age, years	52 (11.8)	69 (10.9)	–17.7 (–21.1 to –14.3)*	69 (11.0)	69 (11.4)
Women	67 (60.4)	21 (30.4)	29.9 (14.2 to 43.6)	18 (32.1)	3 (23.1)
Ethnicity					
European	74 (66.7)	60 (87.0)	–20.3 (–32.0 to –6.6)	48 (85.7)	12 (92.3)
Māori	25 (22.5)	7 (10.1)	12.4 (–0.1 to 23.1)	6 (10.7)	1 (7.7)
Pacific Peoples	3 (2.7)	1 (1.4)	1.3 (–6.5 to 7.0)	1 (1.8)	0 (0.0)
Asian	7 (6.3)	0 (0.0)	6.3 (–1.2 to 13.0)	0 (0.0)	0 (0.0)
Other	2 (1.8)	1 (1.4)	0.4 (–7.2 to 5.7)	1 (1.8)	0 (0.0)
Risk factors and history					
Hypertension	33 (29.7)	42 (60.9)	–31.1 (–45.1 to –15.4)	33 (58.9)	9 (69.2)
Dyslipidaemia	20 (18.0)	29 (42.0)	–24.0 (–38.0 to –9.5)	21 (37.5)	8 (61.5)
Diabetes mellitus	8 (7.2)	5 (7.2)	0.0 (–10.3 to 8.3)	5 (8.9)	0 (0.0)
Current smoker	39 (35.1)	13 (18.8)	16.3 (1.8 to 28.9)	11 (19.6)	2 (15.4)
Family history of premature CAD	36 (32.4)	16 (23.2)	9.2 (–5.4 to 22.4)	11 (19.6)	5 (38.5)
Known ischaemic heart disease	10 (9.0)	32 (46.4)	–37.4 (–50.5 to –23.4)	26 (46.4)	6 (46.2)
Onset of symptoms to presentation, hours	14 (3.9–39.5)	15 (1.4–40.5)	1*†	14 (1.4–38.6)	37 (7.0–48.2)
Proportion of patients presenting <3 hours from symptom onset, hours	20 (18.7)	22 (34.4)	–13.9 (–27.9 to –0.3)	19 (37.3)	3 (23.1)
Proportion of patients presenting ≥3 hours from symptom onset, hours	87 (81.3)	42 (65.6)	17.5 (3.1 to 31.9)	32 (62.7)	10 (76.9)

Data are presented as n (%) or mean (SD) or median (IQR).
*Absolute difference (95% CI) between patients identified as low-risk and non-low-risk.
†Not appropriate to construct CIs; Mann-Whitney U test was p=0.78, indicating it is unlikely that there is a difference in time from symptom onset to presentation between low-risk and non-low-risk groups.
CAD, coronary artery disease.

Table 2 Primary and secondary outcomes

	Low-risk (n=111)	Non-low-risk (n=69)
Patient management		
Low-risk patients managed in general practice	111 (100.0)	0 (0.0)
Low-risk patients managed in general practice per protocol	106 (95.5)	0 (0.0)
Patients managed in rural general practice	111 (100.0)	13 (18.8)
Patients referred to hospital	0 (0.0)	56 (81.2)
MACE diagnosis		
Total patients with MACE at index presentation	0 (0.0)	8 (11.6)
Total patients with MACE during readmissions within 30 days from index presentation	0 (0.0)	2 (2.9)
Total patients with MACE within 30 days of index presentation (from index presentation or readmission)	0 (0.0)	9 (13.0)
ACS diagnosis		
Total patients with ACS at index presentation	0 (0.0)	13 (18.8)
Total patients with an ACS during readmissions within 30 days from index presentation	0 (0.0)	3 (4.3)
Total patients with an ACS within 30 days of index presentation (from index presentation or readmission)	0 (0.0)	15 (21.7)
Non-emergency coronary interventions during index presentation		
PCI	0 (0.0)	4 (5.8)
CABG	0 (0.0)	1 (1.4)

Data are presented as n (%).
One patient had an NSTEMI at presentation and second NSTEMI within 30 days of index presentation.
Diagnoses and coronary interventions are only for the non-low-risk patients referred to hospital.
ACS, acute coronary syndrome; CABG, coronary artery bypass grafting; MACE, major adverse cardiac event; NSTEMI, non-ST-elevation myocardial infarction; PCI, percutaneous coronary intervention.

concentrations or experienced a MACE within 30 days (table 2).

Patients' satisfaction with care

The response rate on the patient satisfaction questionnaire at the participating practices from 31 October 2016 to 30 April 2018 was 75.0% (111 of 148). Of the respondents, 67 (60.4%) were classified and managed as low-risk and 44 (39.6%) were classified as non-low-risk, while 37 (84.1%) were referred to hospital. Overall, 94.0% of low-risk respondents and 95.5% of non-low-risk respondents were satisfied with the service they received at the general practice (table 3). Of the patients identified as low-risk, 94% reported good to excellent satisfaction with care outcomes in the practices using this new model of care.

Table 3 Patient satisfaction with care questionnaire outcomes

	The urgency with which you were assessed?	The thoroughness of your assessment?	Understood explanation of tests and procedures?	Understood explanation of the process and time expectation?	Delivery of the service was more effective than expected?	Overall how satisfied are you with the service you received?
Low-risk patients						
Good–excellent	65 (97.0)	63 (94.0)	62 (92.5)	64 (95.5)	64 (95.5)	63 (94.0)
Fair	1 (1.5)	3 (4.5)	2 (3.0)	0 (0.0)	1 (1.5)	2 (3.0)
Poor	1 (1.5)	1 (1.5)	3 (4.5)	3 (4.5)	2 (3.0)	2 (3.0)
Non-low-risk patients						
Good–excellent	42 (95.5)	43 (97.7)	43 (97.7)	36 (81.8)	42 (95.5)	42 (95.5)
Fair	0 (0.0)	1 (2.3)	1 (2.3)	8 (18.2)	2 (4.5)	1 (2.3)
Poor	2 (4.5)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (2.3)

Data are presented as n (%).

Participating centres' acceptability

The pathway was considered feasible and acceptable by the general practices to the extent that it has been maintained as the standard of care in the participating centres.

Intervention outcomes

Among the 111 (61.7%) patients who were classified as low-risk by the clinical pathway, all were managed in rural general practice with no 30-day MACE (0.0%, 95% CI 0.0% to 3.3%) (table 2). In this pilot evaluation, the sensitivity of the pathway for 30-day MACE was 100.0% (70.1% to 100.0%) and the NPV was 100.0% (96.7% to 100.0%). The specificity and PPV were 63.8% (56.4% to 70.6%) and 12.5% (6.7% to 22.1%), respectively.

30-day MACE in non-low-risk patients

Of the 56 patients classified as non-low-risk and referred to hospital, 9 (16.1%) had at least one MACE within 30 days of presentation. Eight (14.3%) had a non-ST-elevation myocardial infarction (NSTEMI) diagnosis during the index presentation and two (3.6%) had NSTEMI during the 30-day follow-up, one of whom had also experienced NSTEMI at presentation. The overall 30-day MACE rate for all patients presenting to participating rural general practices with suspected cardiac chest pain was 5.0%. The occurrence of 30-day MACE was 13.0% (6.5%–23.8%) for the 69 patients identified as non-low-risk.

30-day ACS and non-emergency coronary revascularisations in non-low-risk patients

An additional six (10.7%) patients identified as non-low-risk were diagnosed with unstable angina, either during the index hospital admission or within the 30-day follow-up. The occurrence of 30-day ACS was 15% (21.7%) for the 69 patients identified as non-low-risk. Non-emergency coronary revascularisation procedures were undertaken in five non-low-risk patients; four had percutaneous coronary intervention and one had coronary artery bypass grafting.

Despite appropriate referral from their general practitioner, one patient classified as non-low-risk by the pathway refused hospital referral due to personal circumstances and self-discharged from the practice. The patient was recalled later that day due to elevated laboratory troponin concentrations, but again declined urgent hospital review. The patient was subsequently admitted to hospital the following day with an NSTEMI.

Of all 69 patients classified as non-low-risk, 56 (81.2%) had an EDACS ≥16, either alone or in combination with positive ECG and/or the presence of red flags (online supplemental table 4). In the majority of patients with an EDACS ≥16, age and gender alone were sufficient to classify them as non-low-risk (78.6%). Occurrence of 30-day MACE according to the results of individual and combination of positive diagnostic test parameters is summarised in online supplemental table 5.

Exercise stress testing

Thirty-three (29.7%) low-risk patients underwent exercise stress testing. These were performed 36 days (IQR 23.0–77.0 days) following index presentation.

POC-cTnI performance

There was good concordance between POC-cTnI concentrations obtained during the pathway assessment and the duplicate hospital or community measured laboratory troponin concentrations (online supplemental table 6). These assays measure different parts of the troponin complex, meaning that concentrations should be correlated rather than the same. Eleven discordant samples were observed. Seven samples from four patients were positive by the POC-cTnI method (range 0.04–0.06 µg/L) but had a laboratory troponin below the relevant cut-off (false positives), as duplicate samples yielded Beckman Coulter Access cTnI concentrations below the upper reference limit (<10 ng/L). Four false negatives were observed with the POC-cTnI assay in the practices. All of these discordant samples had measured concentrations very near the relevant cut-off and no patient with a discordant troponin experienced a MACE within 30 days.

DISCUSSION

In this pilot evaluation of a rural accelerated diagnostic chest pain pathway, over 60% of patients in whom urgent hospital referral would be usual practice were identified as low-risk for AMI and managed in rural general practice without 30-day MACE. This demonstrates that a structured pathway is feasible to implement, and initial results suggest it may be possible to safely and markedly reduce hospital transfer of low-risk patients presenting to rural general practice with suspected cardiac chest pain, negating the requirement for often lengthy transfers to rural or base hospitals for further assessment and serial troponin testing. Patients identified as non-low-risk had a 30-day MACE rate of 13.0% and an ACS rate of 21.7%, with revascularisation undertaken in 5 of 69 patients, thus emphasising the importance of protocol adherence through referral of patients identified as non-low-risk to urgent specialist hospital services. The structured model of care for chest pain management was well accepted by patients, as indicated by the positive feedback from the patient satisfaction-of-care questionnaire. Good agreement was observed between the POC-cTnI concentrations measured as part of the pathway assessment and duplicate laboratory troponin concentrations, providing reassurance about the safety of using these assays as part of an ADP in rural primary care in this pilot.

To our knowledge, this is the first 'real-life' evaluation of an ADP incorporating POC-cTn specifically created for rural use in a general practice setting. The pathway provided reassurance for expedited discharge of low-risk patients presenting with an episode of chest pain suggestive of cardiac ischaemia within 72 hours directly from the practice to home, who would normally have been

referred to hospital for serial cardiac troponin testing and further work-up. The evaluation of this pragmatic implementation was undertaken without mandating the pathway so that a realistic measurement of the impact of introduction could be obtained. There was generally good adherence to the pathway by practice staff, although almost one in five presenting patients who were classified as non-low-risk by the pathway were not referred to hospital, against protocol guidance. The 13 non-low-risk patients who were managed at home had similar cardiovascular risk profiles to those who were referred to hospital (table 2 and online supplemental table 4). While we cannot be sure of the rationale behind the decision not to refer these patients, this finding highlights the importance that clinicians using the pathway appreciate the risks associated with discharging non-low-risk patients without further work-up.

This assessment of the new pathway implementation had several limitations. Although the sensitivity for MACE was 100% in the non-low-risk group, the 95% CI was large, ranging from 70% to 100%. This was a pilot study and not intended to make a precise estimate of safety. Thus, the small sample size limits generalisability of the findings, but does however highlight the feasibility of its implementation in rural practice and provides support for evaluation of the rural pathway in larger studies.

This approach may not be appropriate for all health systems or medicolegal environments. Its efficacy would depend on the rates of presentation of patients with possible ACS to rural general practices and the likelihood that they would be transported to an urban hospital. The proportion we identified as not needing transport to an urban hospital is dependent on the subjective judgement of the attending physicians that the patients they included in the pathway would normally have needed transportation. Therefore, in our evaluation the true proportion of patients who can avoid hospital presentation may be misrepresented by our sample as we cannot rule out that some patients presenting to the practices may have not been recorded in the customised template built into the practice management system and instead were transferred directly to hospital without pathway assessment. Understanding chest pain patient referral patterns would enable us to identify any missed opportunities and have most impact in the community. Although successfully used for AMI rule-out in EDs, the EDACS decision cut-off to identify low-risk patients has not been optimised for use in the rural general practice setting. The prevalence of disease in a patient population influences the performance of a clinical tool. Where the pretest probability for AMI is low, such as in these rural general practices, the application of a clinical tool is likely to produce more false positives. It is possible that the threshold for EDACS may require adjustment to enable the identification of a larger proportion of low-risk patients suitable for management in rural general practice care. This would require validation in larger cohorts of patients presenting with possible AMI to allow the derivation of an optimal threshold in the rural primary care setting. In order to improve the clinical sensitivity of the Abbott POC-cTnI i-STAT assay, a cut-off (40 ng/L) below the manufacturer's recommendation (99th percentile) was employed at the practices, in line with the Australian Academy of Clinical Biochemistry recommendations.[18] This resulted in only two false positive results that resulted in hospital transfer. Overall there was excellent concordance of POCT and central laboratory samples (online supplemental table 6). A previous meta-analysis of pooled individual data of 3099 patients from five studies in different countries was used to derive a clinical prediction rule in primary care to identify those with and without a coronary artery disease (CAD) diagnosis to improve the validity.[27] When this rule was applied in a study population with a CAD prevalence of 13.2%, the probability of CAD was 2.1% (95% CI 1.1% to 3.9%) for patients with a score below the prediction rule cut-off and 43.0% (95% CI 35.8% to 50.4%) when the score was above or equal to the cut-off.[27]

The pilot findings are consistent with urban ED studies in which the same ADP was implemented but using a laboratory troponin assay.[16 17] Introducing the pathway to rural general practices resulted in higher early discharge (to home) rate, compared with urban hospital ED cases with suspected ischaemic chest pain (60% vs 30%–40%).[15] This may reflect a higher proportion of low-risk cases among patients presenting to rural general practice compared with urban EDs. Consistent with this, there was also a lower 30-day MACE rate in rural general practice sites (5% vs 15%).[15 17] Of interest the time from symptom onset (median 15 hours) was longer for these patients than those presenting to an urban ED with similar symptoms (median 3 hours).[16] There is some evidence to suggest that people living rurally defer presentation to health services for a variety of reasons, including geography, availability of services, rural culture and cost of accessing services.[28] Our findings concur with previous studies showing that lowering the cut-off for POC-cTn assays, such as cTnI (i-STAT), improved the clinical sensitivity from 34% to 81%.[19]19 Several high-sensitivity POC troponin assays are now in production or have undergone clinical studies.[29] Enhanced precision and analytical sensitivity of these assays should make it possible for rural general practice to fully adopt ADPs that have already been proven to allow the safe early discharge of similar patients from urban hospitals, while ensuring that patients at high risk of MACE are reliably identified and managed, further improving the efficiency and safety of this pathway. These assays may take several years to be validated and widely available and an evidence-based ADP is required for rural health facilities reliant on current-generation POC-cTn.

The quality improvement demonstrated by this ADP evaluation has important implications for health resources and improved patient outcomes. Such an approach has the potential to reduce travel to regional or base hospitals, reduce unnecessary ED presentations, and provide early reassurance for those identified as low-risk.

Adoption of this diagnostic pathway would allow more rural autonomy, benefiting both patients and secondary and tertiary healthcare services. Furthermore, adding a structured approach to chest pain assessment not only allows identification of low-risk patients who are suitable for management closer to home, but also of those non-low-risk patients who warrant urgent hospital referral and treatment. Given that the pathway was considered feasible and acceptable by the general practices and has been maintained as the standard of care in the participating centres supports the sustainability of this approach. Implementation requires training in use of POC technology, use of ECG, if not already in use in the general practice (as they are in NZ), and in the application of EDACS.

Although the NPV of the ADP for 30-day MACE was 100%, a larger study is required to verify the pilot study findings and allow the NZ Cardiac Network to endorse the use of an ADP using POC-cTn in rural communities into its national policy. A prospective observational evaluation is currently underway to verify the safety and efficacy of the pathway using POC-cTn to identify low-risk patients and allow their early discharge from rural hospitals or directly from general and urgent care practices in NZ.[30] In this we hope to recruit 1000 patients with suspected cardiac chest pain. Safety will be determined by measuring the proportion of 30-day MACE. This larger follow-up evaluation will assist in providing an indication of the potential scalability of the ADP implementation in rural settings.

CONCLUSION

The use of a specifically adapted ADP incorporating POC-cTn in a rural general practice setting was feasible. This pilot implementation demonstrated acceptable clinician adherence and good patient satisfaction with care. Preliminary results suggest that it may possibly be safe and effective at reducing the transfer of low-risk patients to hospital, thereby allowing management in general practice, with no 30-day MACE. Larger prospective evaluations are required to validate the safety and efficacy of this structured clinical pathway for assessment and management of chest pain in rural settings. Adoption of a validated rural accelerated chest pain pathway by NZ general practices has the potential to reduce health service burden and improve patient outcomes by early reassurance for low-risk patients, reduce travel to regional or base hospitals, and improve identification of patients at non-low-risk of AMI.

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REFERENCES

1 Than M, Cullen L, Reid CM, *et al*. A 2-h diagnostic protocol to assess patients with chest pain symptoms in the Asia-Pacific region (aspect): a prospective observational validation study. *Lancet* 2011;377:1077–84.
2 Than M, Cullen L, Aldous S, *et al*. 2-Hour accelerated diagnostic protocol to assess patients with chest pain symptoms using contemporary troponins as the only biomarker: the adapt trial. *J Am Coll Cardiol* 2012;59:2091–8.
3 Hollander JE. The continuing search to identify the very-low-risk chest pain patient. *Acad Emerg Med* 1999;6:979–81.
4 Goodacre S, Cross E, Arnold J, *et al*. The health care burden of acute chest pain. *Heart* 2005;91:229–30.
5 Ellis C, Gamble G, Devlin G. The management of acute coronary syndrome patients across New Zealand in 2012: results of a third comprehensive nationwide audit and observations of current interventional care. *N Z Med J* 2013;126:33.
6 Andersson PO, Karlsson J-E, Landberg E, *et al*. Consequences of high-sensitivity troponin T testing applied in a primary care population with chest pain compared with a commercially available point-of-care troponin T analysis: an observational prospective study. *BMC Res Notes* 2015;8:210.
7 Bösner S, Becker A, Haasenritter J, *et al*. Chest pain in primary care: epidemiology and pre-work-up probabilities. *Eur J Gen Pract* 2009;15:141–6.
8 Roche T, Jennings N, Clifford S, *et al*. Review article: diagnostic accuracy of risk stratification tools for patients with chest pain in the rural emergency department: a systematic review. *Emerg Med Australas* 2016;28:511–24.
9 Pope JH, Aufderheide TP, Ruthazer R, *et al*. Missed diagnoses of acute cardiac ischemia in the emergency department. *N Engl J Med* 2000;342:1163–70.
10 Goodacre S, Thokala P, Carroll C, *et al*. Systematic review, meta-analysis and economic modelling of diagnostic strategies for suspected acute coronary syndrome. *Health Technol Assess* 2013;17:1–188.
11 Cullen L, Greenslade J, Merollini K, *et al*. Cost and outcomes of assessing patients with chest pain in an Australian emergency department. *Med J Aust* 2015;202:427–32.
12 Diercks DB, Roe MT, Chen AY, *et al*. Prolonged emergency department stays of non-ST-segment-elevation myocardial infarction patients are associated with worse adherence to the American College of Cardiology/American heart association guidelines for management and increased adverse events. *Ann Emerg Med* 2007;50:489–96.
13 Sprivulis PC, Da Silva J-A, Jacobs IG, *et al*. The association between hospital overcrowding and mortality among patients admitted via Western Australian emergency departments. *Med J Aust* 2006;184:208–12.
14 Pines JM, Hollander JE. Emergency department crowding is associated with poor care for patients with severe pain. *Ann Emerg Med* 2008;51:1–5.
15 Than M, Aldous S, Lord SJ, *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.
16 Than MP, Pickering JW, Aldous SJ, *et al*. Effectiveness of EDACS versus adapt accelerated diagnostic pathways for chest pain: a pragmatic randomized controlled trial embedded within practice. *Ann Emerg Med* 2016;68:93–102.
17 Than MP, Pickering JW, Dryden JM, *et al*. ICare-ACS (improving care processes for patients with suspected acute coronary syndrome): a study of Cross-System implementation of a national clinical pathway. *Circulation* 2018;137:354–63.
18 Simpson P. Recommendations for Use of Point-of-Care (POC) Troponin Assays in Assessment of Acute Coronary Syndrome [Internet]. *Australasian Association of Clinical Biochemists; 2016 [sighted* 2021 Feb 19]. Available: https://www.aacb.asn.au/resources/recommendations-for-use-of-troponin-poc/recommendations-for-use-of-troponin-poc
19 Schneider HG, Ablitt P, Taylor J. Improved sensitivity of point of care troponin I values using reporting to below the 99 th percentile of normals. *Clin Biochem* 2013;46:979–82.
20 Lilley R, de Graaf B, Kool B, *et al*. Geographical and population disparities in timely access to prehospital and advanced level emergency care in New Zealand: a cross-sectional study. *BMJ Open* 2019;9:e026026.
21 Aldous S, Mark Richards A, George PM, *et al*. Comparison of new point-of-care troponin assay with high sensitivity troponin in diagnosing myocardial infarction. *Int J Cardiol* 2014;177:182–6.
22 Meek R, Braitberg G, Cullen L, *et al*. Outcome at 30 days for low-risk chest pain patients assessed using an accelerated diagnostic pathway in the emergency department. *Emerg Med Australas* 2016;28:279–86.
23 Than M, Flaws D, Sanders S, *et al*. Development and validation of the emergency department assessment of chest pain score and 2 H accelerated diagnostic protocol. *Emerg Med Australas* 2014;26:34–44.
24 Luepker RV, Apple FS, Christenson RH, *et al*. Case definitions for acute coronary heart disease in epidemiology and clinical research studies: a statement from the AHA Council on epidemiology and prevention; AHA statistics Committee; world heart Federation Council on epidemiology and prevention; the European Society of cardiology Working group on epidemiology and prevention; centers for disease control and prevention; and the National heart, lung, and blood Institute. *Circulation* 2003;108:2543–9.
25 Apple FS, Sandoval Y, Jaffe AS, *et al*. Cardiac troponin assays: guide to understanding analytical characteristics and their impact on clinical care. *Clin Chem* 2017;63:73–81.
26 Thygesen K, Alpert JS, Jaffe AS, *et al*. Fourth universal definition of myocardial infarction (2018). *J Am Coll Cardiol* 2018;72:2231–64.
27 , Aerts M, Minalu G, *et al*, International Working Group on Chest Pain in Primary Care (INTERCHEST). Pooled individual patient data from five countries were used to derive a clinical prediction rule for coronary artery disease in primary care. *J Clin Epidemiol* 2017;81:120–8.
28 Ministry of Health. Urban–Rural health comparisons: key results of the 2002/03 New Zealand health survey. Wellington: Ministry of health, 2007. Available: https://www.health.govt.nz/publication/urban-rural-health-comparisons-key- results-2002-03-new-zealand-health-survey [Accessed 2 Feb 2007].
29 Pickering JW, Young JM, George PM, *et al*. Validity of a novel point-of-care troponin assay for Single-Test Rule-Out of acute myocardial infarction. *JAMA Cardiol* 2018;3:1108–12.
30 Miller R, Young J, Nixon G, *et al*. Study protocol for an observational study to evaluate an accelerated chest pain pathway using point-of-care troponin in New Zealand rural and primary care populations. *J Prim Health Care* 2020;12:129.