Use of an Accelerated Diagnostic Pathway allows rapid and safe discharge of 70% of chest pain patients from the Emergency Department

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Abstract

Introduction The majority of patients who present to the Emergency Department (ED) with chest pain, do not have Acute Coronary Syndrome (ACS). Rapid, safe discharge home for this large group is hampered by clinical uncertainty. A pragmatic Accelerated Diagnostic Pathway (ADP) used in our ED achieves this goal.

Aim To demonstrate the safety and utility of a locally developed ADP. The primary outcome for patients who were identified as non-high risk by our ADP was death or acute myocardial infarction (AMI) at 30 days. Secondary outcomes were ED length of stay, discharge rates, provocative testing and revascularisation rates.

Method This is a prospective observational convenience cohort study of chest pain patients presenting to a regional ED excluding ST-elevation myocardial infarction (STEMI). Using a locally derived ADP, patients were classified as high risk or non-high risk for 30-day death or AMI. Patients could be classified as high risk on the basis of ECG change, troponin elevation, or senior clinician “gestalt” irrespective of negative serial ECGs and troponins. All others were classified non-high risk and were followed up at 30 days.

Results There were 452 patient events with the ADP identifying 75% as non-high-risk (93% of these patients were actually discharged). All patients were successfully followed up for 30-day outcomes. The sensitivity and negative predictive value of the ADP was 100% (95% CI: 99–100%). Specificity was 83% (95% CI: 79–87%). The average ED length of stay was 4 hours 5 minutes. There were low rates of revascularisation (1.5%) and provocative testing (6.2%) in the non-high risk group.

Conclusion This ED ADP for chest pain rapidly and safely identified patients who were not at high risk of a short-term AMI or death.
Our hospital uses the 5\textsuperscript{th}-generation high-sensitivity cardiac troponin-T (hs-cTnT) (Roche\textsuperscript{®}); enabling earlier detection of cardiac injury. Mild elevation in troponin is common in patients without AMI (cardiac failure, cardiomyopathy, diabetes, and renal impairment).

There is evidence that delta change (up or down) in hs-cTnT levels in conjunction with an absolute cutoff value for MI (of >52 ng/L) may increase specificity without a significant reduction in sensitivity with the current literature supporting the use of absolute delta values as opposed to relative change.\textsuperscript{21-26}

This ADP, in addition to standard early ECG interpretation, uses very early serial troponins (applying absolute and delta cutoffs) and the “safety-net” of senior doctor gestalt (that is, the presence significant clinical concern for an acute coronary syndrome despite normal investigations).

Our aim was to show that a locally developed and applied ADP can rapidly and safely identify a cohort of chest pain patients who are not at high risk of death or MI within 30 days of ED presentation.

This study was conducted in a regional New Zealand ED (Nelson Hospital). Five percent of our 26,000 annual ED attendances are for chest pain, 18\% of which ultimately have an ACS diagnosis.

The ED physician group consists of five specialists with postgraduate qualification (Fellow of the Australasian College for Emergency Medicine) and three without.

Our hospital has a cardiology service with in-hours access to emergent coronary intervention. An outpatient chest pain clinic provides provocative testing for non-high risk patients and can only be accessed via primary care.

The recruitment period was from August 2013 to April 2014. Recruitment was by convenience sampling as there is no specialist emergency physician directly available in our department from 0000–0800.

Methods

This is a single-centre prospective observational study of a convenience cohort of ED chest pain patients subjected to a local ADP stratifying for risk of AMI or death at 30 days.

Northern B Health and Disability Committee ethics approval was obtained (reference number 13/NTB/72) in June 2013 and was registered with the Australia New Zealand Registry of Clinical Trials ACTRN12614000417684.

We powered the study to show a primary outcome rate of <1\% at the 95\% confidence interval. Our calculations used data from two recent studies and calculation methods for diagnostic testing, published by Jones et al.\textsuperscript{26–28} We determined a minimum number of between 373 and 400.

All patients with a presenting complaint of chest pain being assessed by an ED doctor were eligible for the study. Patients exited the study if given a clear non-cardiac diagnosis during ED workup. This included pulmonary embolism, pneumonia, pericarditis, but specifically excluded musculoskeletal chest pain and gastroesophageal reflux; both of these are well-documented misdiagnoses resulting in missed ACS cases.

Written consent for 30-day follow-up was obtained from eligible patients prior to the availability of troponin result. Those patients not seen by an ED physician primarily were managed under the supervision of an ED physician, including ECG interpretation and final risk assessment/gestalt. Patients with STEMI were followed up, although not included in the study.
Figure 1. ADP pathway

**Chest pain pathway for Nelson ED**

All chest pain patients get immediate ECG interpretation and focused history and offered consent for 30 day follow-up.

- **STEMI pathway**
  - Yes → STEMI?
  - No → ECG changes**

**Patients may be re-stratified or alternatively diagnosed by ED SMO at any point during ED evaluation**

- **ECG changes**
  - Yes → Evaluated as very low risk (<1% 30 day event)
  - No → **ECG Changes are ST elevation not otherwise meeting thrombolysis criteria, ST depression, dynamic ST change (e.g. Wellens) dynamic T waves***

**Enter SMO estimate of probability of 30 day event (unexpected death/ACS) before Trop T result on data sheet**

1. **Troponin time zero**
   - <14ng/L
   - 14-52ng/L
   - >52ng/L

2. **Repeat HS Trop T at 2 hours**
   - 2hr Δ*** ≤5ng/L → Yes
   - Δ>5ng/L or repeat Trop T
     - >52ng/L → Yes → **High risk**
     - ≤52ng/L → No → **Non-high risk**

**SMO, please enter your non-high risk vs high gestalt on data sheet (irrespective of patient disposition)**

*Exclusion criteria

Age <15 yrs
Unable to consent (dementia/psychiatric/behavioural disturbance)
Previously enrolled to this study

***Δ Delta value (either positive or negative)
Patients could be stratified as high risk by one of three ways:

- Ischaemic ECG at any stage during ED admission (irrespective of troponin result).
- Initial troponin >52 ng/L or a 2-hour delta (change up or down) troponin of >5 ng/L.
- If despite these being negative the ED physician had significant clinical concern for ACS at the end of the evaluation. This was included as a “safety net” as there is a well-documented rate of unstable angina with negative ECG/biomarkers.

Patients not meeting any of these criteria were stratified as non-high risk.

We derived our absolute troponin and delta troponin cutoff values from recent work by Reichlin et al. This group used 1-hour delta troponin values. As there were some members of our group not comfortable discharging patients based on 1-hour delta troponins, we elected to conservatively extrapolate the available data to a 2-hour hs-cTnT delta of 5 ng/L. The same paper provided an absolute cutoff off for high risk of 52 ng/L.

A ‘time zero’ hs-cTnT was obtained as close to time of ED triage as possible with a second troponin sample taken (if required) approximately 2 hours later. Patients with duration of pain greater than 6 hours and initial troponin <14 ng/L, were not required to have a second troponin, and were considered troponin negative. Patients felt by the clinician to have a very low risk of ACS (arbitrarily <1%) were not required by the ADP to have biomarker testing.

All non-consented patients were identified by review of daily departmental presenting complaint. This included patients referred directly from general practitioners to the cardiology service, but seen in our department. These patients had their records reviewed at 30 days for evidence of death or MI, but were not included in the study.

The ECG was considered ischemic if there was new or presumed new ST elevation or significant ST depression (>1 mm) or T wave inversion at any stage during ED assessment.

All consented patients were followed up with structured telephone or email interview no sooner than 30 days from their index presentation. Primary outcome events were recorded by one of two investigators. Independent adjudication in unclear cases of AMI was by consultant cardiologists.

Data was collected on a standardised data sheet and entered into Microsoft Excel spreadsheet and statistical analysis was web-based using http://www.medcalc.org/calc/diagnostic_test.php

**Results**

We recorded 852 ED encounters for 726 patients with chest pain potentially consistent with ACS. There were 40 STEMIs; a rate of 4.7% of all chest-pain patients.

Our study cohort consisted of 436 chest pain patients who presented 452 occasions. 60% were male. The mean age was 63±14.5 years (1SD). All consented patients were successfully followed-up.

341 patients were stratified as non-high risk by this ADP, none of who had a 30-day event.
Twelve percent (n=40) of the non-high risk group underwent provocative evaluation (exercise tolerance testing or stress ECHO). Only seven occurred within 72 hours of discharge.

It is worth noting that none of the 131 chest pain patients with a time zero hs-cTnT of 5 ng/L or less experienced a 30-day event. This was 38% of the non-high risk group.

Additionally, 29% of the non-high risk group had a documented history of IHD.

Of the 111 chest pain patients who were considered high risk (HR) by the ADP 44 (40%) had a 30-day primary event (see Table 1).
Table 1. Characteristics of the high-risk group by ADP with 30-day event rates

<table>
<thead>
<tr>
<th>Sub-group</th>
<th>n, (%)</th>
<th>30-day event n=44 (% of sub-group)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic ECG</td>
<td>36 (32%)</td>
<td>17 (47%)</td>
</tr>
<tr>
<td>hs-cTnT ≥52 ng/L</td>
<td>23 (21%)</td>
<td>15 (65%)</td>
</tr>
<tr>
<td>∆ hs-cTnT ≥5 ng/L</td>
<td>22 (26%)</td>
<td>10 (46%)</td>
</tr>
<tr>
<td>Clinical concern</td>
<td>30 (27%)</td>
<td>2 (7%)</td>
</tr>
</tbody>
</table>

113 patients did not have a 2-hour troponin. This was made up of patients with an initial diagnostic troponin (n=23), as well as those with pain onset of more than 6 hours in who had a hs-cTnT <14 (n=90).

Four patients in the non-high risk group had revascularisation (1.2%). Two had recent AMIs prior to their index ED presentation, one of who was already scheduled for a staged LAD stent following a STEMI, one who was a direct referral to Cardiology (opportunistically enrolled to the study) and was thought to have progressive disease requiring stenting.

Of the remaining two, one received semi-elective coronary artery grafts within the 30-day period suffering a perioperative stroke followed by a NSTEMI during rehabilitation. The other had been a electively stented 1 month prior to index ED presentation and went on to have a delayed stent-related coronary artery dissection (29 days following consent for the study), requiring further stenting. By comparison, 23% of the high-risk group underwent revascularisation.

Both sensitivity and negative predictive value for the ADP was 100% (95%CI: 98.9–100% and 92–100% respectively). Specificity and positive predictive values were 83% and 39% respectively.

Overall average ED length of stay was 4 hours 5 minutes. There were 262 patients who presented within the study hours but who were not consented for follow-up. Most of these were either direct referral to the cardiology service, therefore not seen by ED medical staff, or referred from primary care with a known troponin result, and therefore unable to enter the study. Four patients declined consent and three who were initially consented were removed due to presence of exclusion criteria (<25 years old, dementia and previous enrolment within 30 days of index presentation). 148 of these 262 patients were discharged, with no evidence of any 30-day adverse event on review of patient records. Of those admitted there were 45 primary events (39%). None of these events were included in our study cohort.

Discussion

This was a study of a locally developed ADP that rapidly identified a large group of ED chest pain patients as safe for discharge within in a mean ED LOS of 4 hours 5 minutes. To our knowledge this is the first prospective study that has safely discharged home 70% of all ED chest pain presentations. Unlike recently published ADP, we did not use a formal scoring tool such as TIMI or GRACE. This enabled a greater proportion of patients to be classified as non-high risk for 30-day events, without affecting safety.

It is noteworthy that there were a higher proportion of patients with documented IHD in the non-high risk group than the high-risk group. This reinforces the idea that high TIMI or GRACE score does not necessarily translate to short-term risk.

We stratified the groups as high risk and non-high risk in an attempt to remove the uncertainty associated with “intermediate risk” patients, simplifying what has become a very complex decision process. Our ADP asks the simple question—“is this patient at high risk of a major adverse cardiac event in the next 30 days?” If not, patients were discharged home. This did not mean they didn’t have cardiac disease but simply that they were safe to have any further investigations organised by their primary care doctor as an outpatient.
Despite a recent call for a “requiem on unstable angina” there is an incidence of unstable coronary plaque/high grade occlusion with negative biomarkers. We used senior clinician assessment/gestalt to provide an important “safety net” for the group of patients with unstable coronary artery disease and negative biomarkers/ECG. All patients felt to be high risk by this criteria were admitted, two (7%) of who ultimately had a diagnosis of NSTEMI.

Previous studies have typically used a triple composite endpoint of death, AMI and revascularisation. There is however a lack evidence showing benefit for revascularisation in stable coronary artery disease. For this reason we specifically chose patient oriented rather than procedurally oriented outcomes.

We identified the following limitations:

- **Convenience sampling was due to the availability of ED SMO presence for clinical risk assessment.** Patients who presented outside of this period (n=97) were electronically followed-up. Using admission and discharge, as surrogates for high and non-high risk respectively, to the best of our knowledge none who was discharged had an adverse event.

- **Missed recruits.** This was due to a number of factors, but was predominantly made up of patients referred directly by general practitioners to the cardiology service rather than an Emergency Physician. There was higher rate of ACS in this group (17% vs 10% in our cohort), likely due to this referral bias. Discharged patients who were missed for recruitment had their electronic record for local and national events reviewed at 30-days. To the best of our knowledge the primary outcome rate for this group was zero. Although not studied, these patients were likely to have had a similar risk stratification process to the ADP, as use and interpretation of 0 and 2 hour troponins was standard practice in our ED at the time.

- **Validity.** The study was performed at a single secondary hospital, with a relatively small group of emergency physicians in a community with strong primary care follow-up. Our ADP requires external validation in other ED settings.

- **The optimum test characteristics of serial hs-cTnT are yet to be defined.** We used an algorithm based on what we believe to be the best available evidence on this topic. Further studies on hs-cTnT in clinical practice are likely to further refine risk stratification using this test.

- **Dependence of the ADP on the senior ED physician’s unstructured judgment.** This was designed as a safety net for the small number of patients with negative hs-cTnT and ECG but who may nonetheless be clinically very concerning. This was a critical safety feature and is a strength of this ADP.

**Conclusion**

We have demonstrated that an Emergency Department Accelerated Diagnostic Protocol allows rapid and safe discharge of the majority (70%) of chest pain patients, with no significant adverse events at 30 days

This approach is likely to result in significantly shorter ED stays, lower costs, fewer admissions and subsequent risks of unnecessary workup for patients. Multi-centre prospective studies would further demonstrate the safety of this approach.
Competing interests: Nil.

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References


