



## Prospective evaluation of a chest pain pathway at Green Lane Hospital

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### Abstract

**Aims** To prospectively evaluate the efficacy and safety of a chest pain pathway at Green Lane Hospital.

**Methods** Between August 1999 and March 2000, patients with non-traumatic chest discomfort considered to be possibly ischaemic were assessed by history, physical examination, baseline electrocardiographic findings and point of care troponin T tests. Those considered not to be high risk entered the pathway which included repeat cardiac markers and electrocardiograms at 6-8 hours, followed by optional stress testing. The patients were followed for at least one year.

**Results** Of 423 patients with chest discomfort compatible with myocardial ischaemia, 173 were enrolled in the pathway, with 19 later transferred off the pathway for clinical reasons. Of the remaining 154 patients, the median duration of hospital stay was 17.3 hours [IQR 8.2, 25.1] (median of 13.1 hours for those who did not undergo stress testing); 111 (72%) stayed in hospital for less than 24 hours. There were no readmissions within 30 days with an acute coronary syndrome. In the year following discharge, three patients had a myocardial infarction (one of whom later died) and four died of non-cardiac causes. At one year, freedom from cardiac death or non-fatal myocardial infarction was 98% [95%CI 95,100].

**Conclusions** The chest pain pathway facilitated patient triage and patients had high event-free survival.

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Many patients who present acutely to hospital with non-traumatic chest discomfort which may be possibly due to myocardial ischaemia do not have definite evidence of an acute coronary syndrome on arrival, nor an obvious non-cardiac cause of their symptoms. Historically, many of these patients were admitted to hospital for several days for observation and investigation, in order to avoid inappropriate discharge of patients who subsequently were found to have elevated cardiac markers confirming a diagnosis of myocardial infarction. This approach causes a significant burden on hospital systems, as acute chest pain can account for 20-30% of hospital admissions.<sup>1</sup>

History, physical examination and electrocardiography at presentation are conclusive of the diagnosis of an acute coronary syndrome in only a small percentage of patients presenting with chest discomfort.<sup>2</sup> The development of new point of care cardiac markers assays, particularly cardiac troponins, has allowed earlier and more accurate risk stratification of these patients.<sup>3,4</sup>

More recently, strategies have been developed to provide rapid triage pathways for selected patients with chest discomfort, thought to be possibly due to myocardial ischaemia, who were not considered to be at high risk. This approach has generally involved pathways which allow early discharge and thus avoid more prolonged

admission to hospital, with its associated increased costs and inconvenience. Chest pain pathways have allowed the rapid triage of low risk patients as well as the earlier identification of patients who may benefit from more aggressive therapy.<sup>5</sup> However, there is little local data regarding the effectiveness and safety of these rapid triage chest pain pathways. Thus, we prospectively evaluated our chest pain triage pathway at Green Lane Hospital.

## Methods

From 24 August 1999 to 27 March 2000, patients who presented acutely to Green Lane Hospital with non-traumatic chest discomfort, thought to be possibly due to acute myocardial ischaemia, were prospectively evaluated for the rapid triage chest pain pathway in the Coronary Care Unit. All patients were assessed on admission using a triage data form, which included past and present history, physical examination, electrocardiographic findings and cardiac marker levels. Patients were admitted directly to the Coronary Care Unit if they were eligible for reperfusion therapy with ST elevation or left bundle branch block not known to be old,<sup>6</sup> or had  $\geq 0.5$ mV ST depression on any electrocardiogram.<sup>7</sup> Patient enrolment in the pathway was based on history, physical examination findings, presentation point of care troponin T level  $< 0.10$   $\mu\text{g/L}$  and the absence of any of the above features on any electrocardiogram following presentation. Other than those aged  $> 75$  years, patients were not enrolled in the pathway if they had any high risk features as defined by the ACC/AHA Guidelines for Unstable Angina and Non-ST segment Elevation Myocardial Infarction.<sup>8</sup> Isolated T wave inversion was not an exclusion criteria. The Auckland Ethics Committee were approached regarding this project, and agreed that this was an audit and therefore written informed consent was not necessary.

Bedside troponin T assay was performed using the CARDIAC reader quantitative assay from Roche Diagnostics [Mannheim, Germany]. The diagnostic criteria for myocardial infarction during the study were: creatine kinase (CK)  $\geq 220$ U/L for males and  $\geq 180$ U/L for females; CK MB mass  $\geq 5$  $\mu\text{g/L}$ ; troponin T  $\geq 0.10$  $\mu\text{g/L}$  (the new discrimination level for myocardial infarction for troponin T is  $0.03$  $\mu\text{g/L}$ ).<sup>9</sup>

All patients were given 300mg of aspirin on admission, unless contraindicated. Either low molecular weight heparin or unfractionated heparin were given if the clinician felt it was indicated. The patients enrolled in the chest pain pathway were observed by experienced coronary care unit nurses for recurrence of symptomatic ischaemia, were given pain relief and reassurance and underwent continuous ST segment monitoring for silent ischaemia and repeat 12-lead electrocardiograms were performed if they had recurrent pain. After an observation period of 6-8 hours, repeat cardiac markers (troponin T, CK and CK MB) and 12-lead electrocardiograms were performed. Patients with normal cardiac marker levels and no electrocardiographic changes were then considered for discharge. Stress testing by exercise tolerance test or stress echocardiography was performed if thought appropriate by the clinical team. Prior to discharge, patient's risk factors were assessed and education initiated where appropriate.

The patients were then followed up for at least one year after their discharge, using direct patient contact, the New Zealand National Health Index (NHI) database and information from general practitioners. Outcomes included transfer off the chest pain triage pathway into the Coronary Care Unit, length of hospital stay, all readmissions, myocardial infarction and death within one year. The patients were stratified according to their prior history of coronary heart disease (CHD). Patients with a history of prior myocardial infarction, percutaneous intervention (PCI), coronary artery bypass grafting (CABG) or abnormal angiogram (demonstrating lesions of greater than 50% diameter loss) were included in the group of "Prior CHD". Patients with a clinical history of angina, including risk factors, but no objective evidence of coronary heart disease were not included ( $n=4$ ). "Smokers" were defined as patients currently smoking at the time of admission; "hypertension" as patients with a history of hypertension, whether on treatment or not, or hypertensive at the time of admission; diabetes as patients on insulin or taking oral hypoglycaemic agents; "hypercholesterolaemia" as a total cholesterol of  $> 5.2$  mmol/L on admission.

**Statistical analysis.** Comparisons between groups for continuous variables were made using unpaired t-test or Mann-Whitney U-test where the distribution of the variable was skewed and the  $\chi^2$  test for categorical variables. Survival curves were constructed using the Kaplan-Meier estimator and differences tested using the log-rank test. The statistical software package SAS release 8.01 was used for all analyses.

## Results

During the study period, 423 patients with non-traumatic chest discomfort thought to be due to myocardial ischaemia were evaluated (Figure 1). Of those, 250 were initially not deemed suitable for the rapid triage chest pain pathway and were admitted directly to the Coronary Care Unit. Of the 173 patients who entered the pathway, 19 (11%) were subsequently transferred to the Coronary Care Unit with positive cardiac markers (11 patients), later electrocardiographic changes (2 patients), ongoing chest pain (2 patients), significantly positive stress tests (2 patients), change in nature of pain consistent with aortic dissection (1 patient) and a clinical decision to perform angiography (1 patient).

**Table 1. Baseline characteristics of patients entering the chest pain pathway and those directly admitted to coronary care.**

	Chest pain pathway n = 173	Coronary care unit n = 250	P-value
Age, years; Mean(SD)	55.9 (15.1)	69.3 (12.8)	<0.001
Gender, male	106 (61%)	147 (59%)	0.56
Smoker	43 (25%)	30 (12%)	0.001
Diabetes	15 (9%)	43 (17%)	0.013
Hypertension	69 (40%)	131 (52%)	0.013
Hypercholesterolaemia	72 (42%)	93 (37%)	0.36
Previous history;			
Angina	50 (31%)	152 (61%)	<0.001
MI	26 (15%)	86 (34%)	0.044
Angiogram	40 (23%)	95 (38%)	<0.001
PCI	16 (9%)	42 (17%)	0.028
CABG	22 (13%)	42 (17%)	0.26
Prior CHD	59 (34%)	171 (68%)	0.001

Chest pain pathway = patients admitted to the chest pain pathway.

Coronary care unit = patients admitted directly to the Coronary Care Unit.

MI = Myocardial infarction.

PCI = Percutaneous coronary intervention.

CABG = Coronary artery bypass grafting.

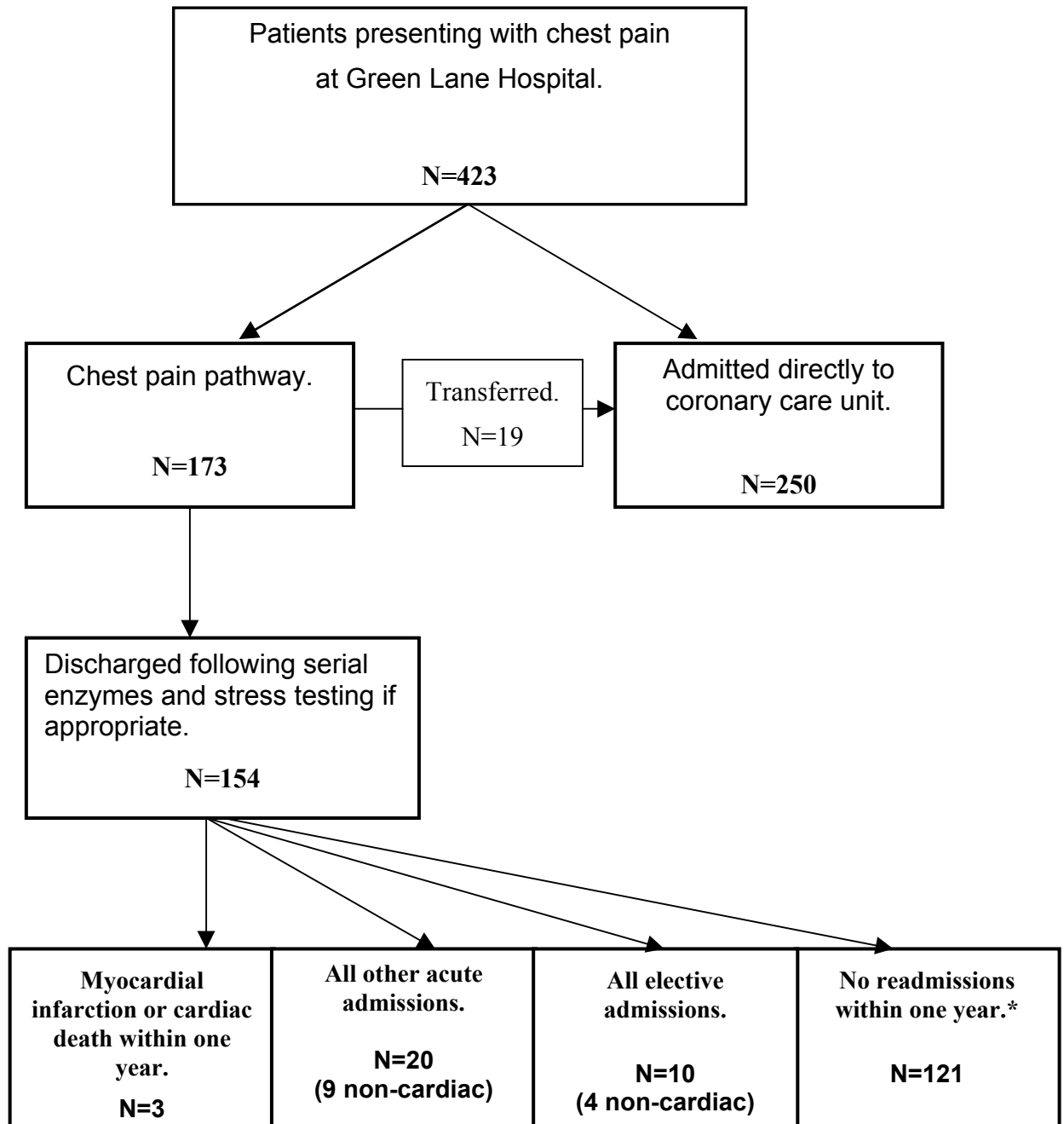
Prior CHD includes patients with MI/PCI/CABG/abnormal angiogram.

The baseline characteristics of patients entering the pathway are shown in Table 1. This group was younger, with a median age of 55.9 years compared with 69.3 years ( $P<0.001$ ) for the patients admitted directly to the Coronary Care Unit. Patients admitted directly to the Coronary Care Unit were more likely to have a history of prior coronary heart disease (68% vs. 34%,  $P<0.05$ ). Of the patients entering the chest pain pathway, the median symptom onset to presentation time was 3.1 hours [IQR 1.7,7.6]. There were no significant differences in the baseline characteristics of the 19 patients who were transferred from the chest pain triage pathway to the Coronary Care Unit compared with those who remained in the pathway.

**Duration of hospital stay.** Median length of hospital stay was 17.3 hours [IQR 8.2, 25.1] and 43 (28%) patients stayed for longer than 24 hours. The median length of stay for those patients who did not undergo in-patient stress testing was 13.1 hours [IQR 5.4,21.5], compared with 21.9 hours [IQR 12.9,26.1] for those who did ( $p=0.038$ ). The presence of significant medical co-morbidities accounted for 23% of

patients staying in hospital for more than 24 hours. Overall, of the 154 patients following the chest pain pathway, 61 (40%) underwent inpatient stress testing; 2 patients underwent stress echocardiography with the remainder performing treadmill exercise tolerance tests. An additional 7 patients underwent urgent outpatient stress testing, which were performed 1 to 10 days after discharge. On reviewing the data forms and laboratory results, no patient was discharged from the chest pain pathway inappropriately with elevated levels of cardiac markers.

**Figure 1. Flow diagram of patients presenting with chest pain.**



\* Vital status of 2 patients was not determined; these 2 patients had no readmission/death recorded.

**One year outcomes.** Vital status was determined in 99% (152/154) of patients at one year. During the follow up period, 4 patients died of non cardiac causes and 3 patients

suffered myocardial infarctions, at 23, 32 and 48 weeks. Of these 3 patients who suffered myocardial infarctions, 2 had previously documented coronary heart disease prior to enrolment in the pathway and had subsequent readmissions with chest pain prior to presenting a third time with myocardial infarction. The third patient, admitted with a fatal myocardial infarction at 48 weeks, was a 89 year old female, who was not stress tested prior to discharge due to her medical co-morbidities. Of the 23 patients who were readmitted to hospital acutely, 3 were readmitted with myocardial infarction, 6 with unstable angina, 2 with unspecified chest pain, 3 with arrhythmias and 9 with non cardiac causes (Table 2, Figure 1). Overall at one year, freedom from cardiac death or non-fatal MI was 98% [95%CI 95,100], with no difference in the event rate for those who had a prior history of coronary artery disease.

**Table 2. One year outcomes following discharge from the chest pain pathway.**

<b>Outcome.</b>	<b>Total number of events</b>	<b>Events in patients with prior CHD</b>
<b>Out of hospital cardiac death*</b>	0	0
<b>Acute admissions</b>		
Fatal myocardial infarction.	1	0
Non-fatal myocardial infarction.	2	2
Unstable angina.	6	5
Arrhythmias.	3	2
Non cardiac chest pain (unspecified).	2	0
Death from non cardiac cause.	4	2
Non-fatal non cardiac readmission.	5	2
<b>Elective admissions</b>		
Cardiac investigations	6	3
Non cardiac cause.	4	1

Prior CHD includes patients with myocardial infarction/percutaneous coronary intervention/coronary artery bypass grafting/abnormal angiogram.

\*There were no out of hospital non cardiac deaths.

## Discussion

In an earlier era a significant minority (2-6%) of patients with chest pain were subsequently found after discharge from hospital to have had a myocardial infarction when the results of later cardiac marker levels were available.<sup>10,11</sup> These patients had a significantly higher mortality than those who were admitted,<sup>12</sup> and to avoid this risk many individuals who at admission had no electrocardiographic changes or elevated levels of cardiac markers were admitted to hospital for several days. A major impetus for the development of chest pain pathways has been to lower costs by the early discharge of individuals who are considered not to be at high risk of death or myocardial infarction and who have normal levels of cardiac markers after several hours of observation.

In our study no patients were discharged with elevated cardiac marker levels and there were no acute coronary events during the first 30 days follow up. Three myocardial infarctions including one cardiac death occurred during the subsequent year. Previous studies of chest pain pathways have demonstrated similar findings of low event rates in those discharged following a period of evaluation. In a study from the Mayo

clinic,<sup>13</sup> 97 patients were discharged directly from the chest pain observation unit, and this group had no primary events in the 6 month follow up period. However, of the 212 patients assigned to the chest pain observation unit in that study, over half were hospitalised and all underwent stress testing prior to discharge. In a chest pain unit study from Amsterdam,<sup>14</sup> the rate of non fatal myocardial infarction or cardiac death in a 1996 cohort was slightly higher than ours with 2.8% at 6 months and 5.7% at 2 years.

From August 1999-March 2000 we used the diagnostic criteria for myocardial infarction for troponin T of  $\geq 0.10\mu\text{g/L}$ . The new diagnostic criteria for myocardial infarction for cardiac troponin T uses a discrimination level of  $0.03\mu\text{g/L}$ .<sup>9</sup> Troponin T may be the only marker required if utilised in a chest pain pathway with discharge decisions being undertaken following a 6-8 hour observation period.<sup>15-17</sup> Of the 5 patients who were discharged from the chest pain pathway who had troponin T levels  $\geq 0.03\mu\text{g/L}$  but  $< 0.10\mu\text{g/L}$ , one was subsequently readmitted 15 weeks later with probable ischaemic chest pain without evidence of myocardial infarction, and one was readmitted 17 weeks later in complete heart block; the remainder had no events within the one year follow up.

The use of multiple point of care cardiac marker assays has recently been reported in studies of patients with probable acute coronary syndromes.<sup>4,18,19</sup> These studies have included patients with ST depression on their presenting electrocardiogram, whereas our study excluded patients with  $\geq 0.5\text{mV}$  ST depression. The presence and extent of ST depression  $\geq 0.5\text{mV}$  has been shown to adversely influence late survival.<sup>7</sup> The Checkmate study<sup>4</sup> assessed a multi-marker strategy, using CK MB, troponin I and myoglobin, and demonstrated that serial testing using 2 markers (CK-MB and troponin I) risk stratified for 30 day mortality better than the use of a single marker. The addition of myoglobin identified patients earlier, and also identified one extra patient (out of 1005 patients in total) at risk of death. This however decreased specificity, with 35 additional patients identified with elevated myoglobin levels ( $>105\mu\text{g/L}$ ). Another study<sup>18</sup> aimed to exclude myocardial infarction, defined as an elevation in CK MB during a 9 hour observation period, within 3 hours of presentation. Using a combination of myoglobin and troponin I levels, a sensitivity and negative predictive value of 96.9% and 99.6% respectively was demonstrated at 90 minutes. These results were not improved by adding CK-MB testing to the samples, nor by additional blood testing at 3 hours. In this study, the median time to presentation was 3.9 hours, which is longer than in our study (median 3.1 hours). In the SMARTT trial,<sup>19</sup> in patients with a median time to presentation of 2.2 hours, a combination of CK-MB and myoglobin on admission and at 60 minutes gave a sensitivity and specificity for the diagnosis of myocardial infarction, based on the World Health Organisation criteria of an elevation of CK-MB in a characteristic pattern or the evolution of electrocardiographic changes, of only 72% and 88.5% respectively.

In the current study point of care testing for troponin T was used at presentation, followed by laboratory assays for troponin T and CK MB mass at 6-8 hours. Point of care testing is recommended when hospital logistics cannot consistently deliver laboratory cardiac marker results within 1 hour.<sup>20</sup> However, the discrimination level for myocardial infarction of the point of care assay for troponin T used in our study was  $0.1\mu\text{g/L}$ , and therefore it did not detect myocardial infarction occurring with troponin T levels of  $0.03\mu\text{g/L}$  to  $0.09\mu\text{g/L}$ , although the current point of care assay

can determine troponin T levels at 0.03µg/L. When this study was conducted we used laboratory troponin T assays as the criteria for discharging patients presenting with non-traumatic chest pain after 6-8 hours of observation.

The use of a rapid triage chest pain pathway has allowed the length of hospital stay to be shortened considerably, compared to historical controls, with approximately 75% of patients being discharged in less than 24 hours in our study. Comparison of lengths of stay in our chest pain pathway compared with other studies<sup>13,14</sup> is confounded by the lack of description of the length of stay in the subgroups of patients who were discharged from other chest pain units/pathways. In these studies<sup>13,14</sup> the median lengths of stay in the emergency department chest pain units were 9-10 hours but a substantial proportion of these patients were admitted, and perhaps these latter patients had shorter lengths of stay within the chest pain units.

At Green Lane Hospital, patients who waited for in-patient stress testing had significantly longer hospital stays (21.9 hours vs 13.1 hours) than those who were discharged without stress testing. In the study from the Mayo clinic, all 72% of patients who entered the chest pain unit and did not develop criteria for admission underwent stress testing (treadmill or perfusion stress testing), which was available daily between 7.00 am and 10.30 pm, including weekends. It is likely that overall hospital length of stay in our study could have been reduced by improved access to stress testing, for example by using appropriately trained Coronary Care Unit staff to perform exercise treadmill testing in the evenings or at weekends.

The use of exercise testing as part of a chest pain pathway has been extensively studied, and has been shown to be both safe and efficacious.<sup>21,22</sup> In a low risk population without electrocardiographic changes, however, the post-test probability of disease is only modestly increased by a positive test. It remains to be determined whether there is marginal utility in performing immediate stress testing or perfusion scanning in all patients in this population with no electrocardiographic changes.

A limitation of our study is that not all patients were followed up early after discharge, whereas previous studies of chest pain units have requested that patients return for a 12-lead electrocardiograph and repeat cardiac marker levels 24 hours following discharge. While all cardiac marker results were reviewed at the time of the audit we did not formally review all electrocardiograms. The baseline characteristics of the patients in our cohort were similar to those in previous reports, except for the exclusion of patients with ST depression.<sup>4,18,19</sup> Though we have previously reported a 20% one year mortality in patients with ST segment depression of  $\geq 0.5$ mm admitted to Green Lane Hospital CCU in 1993<sup>7</sup> we did not perform follow-up of the 250 patients with probable acute coronary syndromes admitted in the current cohort. Also we did not perform cost-effectiveness analyses.

We conclude that the rapid triage chest pain pathway at Green Lane Hospital was safe, effective and reduced admission times. The safety of the chest pain triage pathway in this population suggests that it could be utilised in the group of patients with chest discomfort presenting to general hospitals, without ST segment deviation on the presentation electrocardiogram.

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