

Near-patient testing for cardiac troponin I to reduce hospital stay in patients presenting with chest pain

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Abstract

Minimising the in-hospital stay of patients with chest pain, within safe limits, is crucial in reducing the cost of health care. The aim of this study was to determine whether the use of near-patient testing for cardiac troponin I could reduce the duration of in-hospital stay for patients presenting with chest pain who were considered to be at low risk of death or myocardial infarction.

This prospective observational study of consecutive patients admitted with chest pain of possible cardiac origin was conducted in a medium-sized district general hospital. A near-patient system for troponin I analysis was compared to traditional laboratory-based troponin I analysis to assess any effect on duration of in-hospital stay in low-risk chest pain patients. Of the 295 patients enrolled in the study, 191 (68.7%) were troponin-negative and were classified as having chest pain of non-cardiac origin or cardiac pain at low risk of major adverse events. The introduction of near-patient testing for cardiac troponin I reduced the mean duration of hospital stay from 30.04 hours to 17.10 hours ($p < 0.001$). At 30-day follow-up no deaths or myocardial infarctions had occurred.

Key words: chest pain, troponin I, cardiac ischaemia, hospital stay.

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Introduction

Chest pain is a frequent cause of hospitalisation and forms a significant proportion of the acute medical workload. Two-thirds of

these cases are found to be not of cardiac origin,¹ one-third to one-half have a probability of acute myocardial infarction (AMI) that is low but not sufficiently low to permit discharge from the emergency department,² and more than 40% of these patients require hospital admission. The inadequacy of current techniques for evaluation and triage of these patients has led to high-cost and low-efficiency clinical practice, in which 2% to 10% of patients with AMI are discharged from the emergency department. Unfortunately, these patients have a mortality rate almost twice that of those admitted to hospital in the first place, and inappropriate discharge of patients with chest pain from accident and emergency departments often features in medical malpractice settlements.³

Minimising the in-hospital stay of patients with chest pain, within safe limits, is crucial in reducing the cost of health care. In one study of 771 patients with chest pain, only three (0.4%) died after primary cardiac arrest. All of these arrests occurred 3–5 days after admission, and the study concluded that a 12-hour (instead of 24-hour) period of observation is normally sufficient to exclude AMI.⁴ These factors, along with the emergence of effective treatments, have spurred efforts to diagnose acute cardiac ischaemia quickly in accident and emergency and medical admission wards.

The traditional approach to ruling out myocardial infarction has relied upon the use of serial electrocardiography which is deemed negative for myocardial ischaemia and upon the absence of evidence of myocardial necrosis as assessed with biochemical markers of cardiac injury. The measurement of biochemical markers of cardiac injury is usually undertaken by a central laboratory. This process inevitably involves a series of 'handoffs' in the delivery of the sample to the laboratory, sample analysis and reporting of results to clinical staff. The net effect is likely to delay patient discharge and extend the length of stay in hospital.

The measurement of cardiac-specific serum troponins has become the mainstay in the biochemical diagnosis of cardiac injury. The absence of an elevation in serum troponin 12 hours or more after an episode of chest pain excludes myocardial infarction and predicts a very low risk of short-term adverse events. More recently, bedside assays for these molecules have become available. We undertook a study to determine whether the use of near-patient testing for serum troponin could reduce duration of hospital stay in patients with chest pain who were considered to be at low risk of myocardial infarction.

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Materials and methods

Lincoln County Hospital is a medium-sized district general hospital in a predominantly rural area. Prior to November 2001, patients with suspected AMI were admitted to the accident and emergency (A&E) department or the medical admissions ward (MEAU). Serum troponin was measured in an on-site biochemistry laboratory.

In an effort to improve 'door-to-needle times' for thrombolytic therapy in patients with AMI, the establishment of a single point of access for patients with suspected AMI was agreed after a redesign. A chest pain assessment unit (CPAU) consisting of two dedicated siderooms was sited within the cardiology ward to allow rapid assessment of suspected AMI and subsequent 'nurse-led' thrombolysis. Patients triaged to be 'low risk' on the CPAU were moved to the MEAU for further medical assessment. Near-patient testing for serum troponin I (Biosite Triage, US) was made available on the CPAU and MEAU to facilitate early discharge and maintain bed availability.

The study was conducted in the MEAU and CPAU of Lincoln County Hospital during the period of July-September 2001 and November 2001-January 2002, in other words before and after the advent of the CPAU.

The 'non-CPAU group' in this study consisted of patients coming to hospital with chest pain of possible cardiac origin who were admitted to MEAU through A&E or directly by the general practitioner. Patients were generally assessed by a Medical Senior House Officer (SHO) and seen by an on-call consultant physician on the MEAU on twice-daily ward rounds. A blood sample for laboratory assay of serum troponin I was recommended 12 hours or more after symptom onset. Clinical guidelines recommend consideration of early discharge if this is normal.

These patients were compared with the patients in the 'CPAU group'. In this group, initial assessment was performed by a CPAU nurse and the patient was subsequently transferred to MEAU where appropriate. A blood sample for near-patient assay of troponin I was recommended 12 hours or more after symptom onset. Clinical guidelines recommend consideration of early discharge if this is normal.

During both study periods, patients were identified during their admission by a research nurse, who prospectively recorded times of admission and of discharge, the final diagnosis and any subsequent readmissions within 30 days of discharge.

Results

There were 295 consecutive patients eligible for the study during this recruitment period. The patients' mean age was 68.1 years (range 22–101 years); 179 (60.7%) were men and 116 (39.3%) were women. Among these patients, 191 were troponin I negative and 104 were troponin I positive. Of the 191 patients who were troponin I negative, a minority (37; 12.5% of the total) were given a diagnosis of possible angina or were already known to have ischaemic heart disease. Table 1 shows the four groups of diagnoses.

The average duration of in-hospital stay for all 191 patients in whom the test was negative in the study period was 24.20

Table 1. Diagnoses for the patients in this study (n=295)

Chest pain cause uncertain	98 (33.2%)
Angina/possible angina	54 (18.3%)
MI/unstable angina	87 (29.5%)
Others	56 (19%)

Key: MI = myocardial infarction

hours. Patients admitted to the MEAU directly or via A&E in the first study period (laboratory testing for troponin I) had an average duration of in-patient admission of 30.04 hours. Patients admitted directly to the CPAU in the second study period (near-patient testing for troponin I) had a reduced average duration of in-hospital stay of 17.10 hours ($p<0.001$).

The medical notes of the patients who were triaged to be low risk and discharged within 24 hours of admission were later reviewed to determine any readmissions. Five patients (three in the non-CPAU group and two in the CPAU group) were readmitted within a 30-day period. In the non-CPAU group, one patient presented with AMI and subsequently died; one presented with unstable angina and was referred for in-patient angiography; and one presented with unstable angina, myocardial infarction was ruled out and she was sent home with an out-patient follow-up appointment. In the CPAU group, two were readmitted; both had MI ruled out and an out-patient exercise tolerance test (ETT) was arranged for one.

Approximately 475 test strips were used on the 295 patients within the study period.

Discussion

'Chest pain - ? cardiac' is a common presentation to coronary care and medical admission units and forms a large part of acute medical workload. These patients occupy a considerable number of bed days and because of the volume of these admissions any intervention which helps to reduce the length of stay would be of great value.

As a result of its improved sensitivity and specificity for myocardial cell necrosis, testing for cardiac troponin has become standard practice in the assessment of such patients. It is generally undertaken in a centralised laboratory, requiring blood samples to be taken and then transported to a remote laboratory and often assayed in batches. Results may be telephoned to the clinical team or made available on a computer system, a time-consuming process with inevitable delays. Most physicians are reluctant to discharge patients home without a negative cardiac troponin result. In contrast, near-patient testing for cardiac troponin can be performed in the clinical area where the patient is situated. Results are available in 15 minutes and can be communicated easily to the clinical team.

Our study shows that the replacement of traditional laboratory-based testing for cardiac troponin with a near-patient test is associated with a significant and sizeable reduction in duration

of stay for patients presenting with chest pain who are deemed to be at low risk of major cardiac adverse events. The mean reduction of stay in our study was almost 13 hours, a major efficiency saving for many hospitals. Moreover, our data suggest the early discharge of low-risk chest pain patients triaged with the aid of near-patient troponin assay is safe.

It is important to emphasise that the comprehensive assessment of these patients extends beyond analysis of biochemical markers of cardiac injury. All patients in this study who were triaged as low risk and suitable for early discharge had been free from chest pain since admission and were without dynamic or evolving ECG changes. The early use of treadmill exercise testing may add further information to risk-stratify the apparently low-risk patient.⁵ A negative troponin assay will not exclude other serious non-cardiac causes of chest pain such as pulmonary embolism and aortic dissection.

There are resource implications in the use of near-patient testing for cardiac troponin. A laboratory-based assay for cardiac troponin may cost £4 per sample (Dr G Griffiths, personal observation) in comparison to a near-patient assay cost of £15 per sample. There may also be costs in association with the purchase of the assay station. However, the reduction in length of stay that we have demonstrated would be expected to more than offset these costs.

There are other methods available to assess and triage patients presenting with 'chest pain - ? cardiac'. ECG without⁶ or with⁷ history, serial measurements of cardiac biomarkers,⁸ the combination of these and ETT,⁹ a recently designed computer-based decision support system (DSP),¹⁰ standard graded exercise testing or stress echocardiography^{11,12} and myocardial perfusion imaging with technetium-99m sestamibi¹³ have all been described with variable additional benefits. However, they may have major resource implications and are unlikely to be realistic options within the UK health system.

The reduction in length of stay seen in our study occurred in association with a redesign of the management of chest pain patients. We cannot exclude the possibility that this might have contributed to the improvements we have described. However, there was no change in consultant ward round frequency or junior medical staff numbers and we believe that the principal factor was the ready availability of results for cardiac markers and the reduction in handoffs and delays promoted by near-patient testing. It may be possible to optimise handling of cardiac troponins within a central laboratory to achieve some of the gains we have described, though the necessarily more complex process would be expected to create delays and seems unlikely to be as time-efficient as near-patient testing.

Conclusion

The use of near-patient testing for cardiac troponin I can reduce the duration of hospital stay in patients presenting with chest pain who are deemed to be at low risk of death or myocardial infarction.



Key messages

- Chest pain is a common presentation to the medical admissions unit
- Troponin I is a marker of cardiac ischaemia with high sensitivity and specificity
- Low-risk patients could be discharged from hospital safely if troponin I (TnI) is negative
- The use of near-patient assay for TnI could reduce the duration of in-hospital stay

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Conflict of interest

None declared.

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