Prospective audit of incidence of prognostically important myocardial damage in patients discharged from emergency department

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Abstract

Objective To assess the incidence of prognostically important myocardial damage in patients with chest pain discharged from the emergency department.
Design Prospective observational study.
Setting District general hospital emergency department.
Participants 110 patients presenting with chest pain of unknown cause who were subsequently discharged home after cardiac causes of chest pain were ruled out by clinical and electrocardiographic investigation.
Interventions Patients were reviewed 12-48 hours after presentation by repeat electrocardiography and measurement of cardiac troponin T.
Main outcome measures Incidence of missed myocardial damage.
Results Eight (7%) patients had detectable cardiac troponin T on review and seven had concentrations ≥0.1 µg/l. The repeat electrocardiogram showed no abnormality in any patient.
Conclusion 6% of the patients discharged from the emergency department had missed prognostically important myocardial damage. Follow up measurement of cardiac troponin T allows convenient audit of clinical performance in the emergency department.

Introduction

Patients presenting to the emergency department with chest pain of unknown cause are a management challenge. Clinical history and examination are imperfect tools for diagnosis. Electrocardiography is the first test, but rigorous comparison based on postmortem diagnosis shows that its diagnostic sensitivity is only 41-61%.[1 2] The admission electrocardiogram, although excellent for selecting patients for thrombolysis,[3] has a diagnostic sensitivity for acute myocardial infarction of 55-75%.[4 5]

Measurement of cardiac troponin T and cardiac troponin I concentrations has greatly improved diagnosis of patients presenting with suspected acute coronary syndromes. The diagnostic time window of these markers is wide, being up to 72 hours; the markers have 100% sensitivity for diagnosing acute myocardial infarction 12 hours after presentation to hospital, and concentrations remain raised for as long as 10 days.[6] Raised concentrations of cardiac troponin T and cardiac troponin I are completely cardiac specific, unlike increases in concentrations of creatine kinase or its MB isoenzyme, which can be due to non-cardiac sources.[7] Detection is diagnostic of myocardial damage in patients admitted with suspected acute coronary syndromes and indicates an unfavourable outcome.[8 9]

We measured cardiac troponin T to assess the incidence of missed myocardial damage in patients presenting with chest pain and suspected acute coronary syndromes discharged from a hospital emergency department.

Participants and methods

We studied patients sequentially attending the emergency department over four months with chest pain. All patients had a full history and clinical examination, a 12 lead electrocardiogram recorded, and an initial blood sample taken for measurement of urea, electrolytes, blood glucose, and creatine kinase concentrations. Cardiac troponin T was not measured at presentation as its diagnostic sensitivity for acute myocardial infarction is equivalent to that of creatine kinase on first presentation with chest pain. Patients were then divided into the following categories: those with definite acute myocardial infarction requiring thrombolysis and admission to the cardiac care unit; those with suspected acute coronary syndromes on clinical or electrocardiographic grounds or with a raised creatine kinase concentration and who required medical referral and possible hospital admission; those in whom acute coronary syndromes were ruled out on clinical and electrocardiographic criteria and who could be discharged from the emergency department; and patients with a definite source of non-cardiac chest pain (obvious musculoskeletal trauma, migraine, chest pain relieved by antacids).

All patients in whom suspected acute coronary syndromes were ruled out and who would not otherwise have been medically reviewed were invited to reattend at 0900 the next working day (if this was 12 to 48 hours after first presentation to the emergency department) for a follow up assessment by a member of the emergency department staff (SP). All patients who accepted were examined again, and a follow up 12 lead electrocardiogram was recorded. A single blood sample was taken by using a 4 ml serum separator gel containing Vacutainer (Becton-Dickinson, Oxford) and sent to the laboratory for measurement of cardiac troponin T concentration. The sample was allowed to clot before spinning, and the serum was then separated and stored at 4°C. Serum was analysed for cardiac troponin T by enzyme linked immunoabsorbent assay (ELISA, Roche Diagnostics, Lewes) as previously described.[6]

Results

During the study 676 patients attended the emergency department with a presenting complaint of chest pain of unknown cause. A total of 268 (40%) patients were admitted for exclusion of acute myocardial infarction and 408 (60%) were discharged. Of the patients

Table 1 Cardiac troponin concentrations in patients discharged from emergency department

Patient	Troponin T (μg/l)
1	0.07
2	0.1
3	0.14
4	0.25
5	0.62
6	0.83
7	1.14
8	3.21

discharged, 122 (30%) were found to have definite non-cardiac chest pain (musculoskeletal injury, chest trauma, or gastrointestinal symptoms) or did not have chest pain on review (migraine, head injury, or upper body laceration). Seventy one (10.5%) were known to have ischaemic heart disease and referred for urgent outpatient medical follow up.

Two hundred and fifteen patients (53%) had a discharge diagnosis of chest pain of unknown cause. Fifty five of these patients were referred for subsequent medical assessment as an outpatient, and 160 were discharged without planned follow up. Of these 160, 110 patients (75 men, 35 women, age range 22.6-88.7 years, median 50.4, interquartile range 41.7 to 63.2) agreed to participate in the study. Fifty patients either declined review or could not be seen within 48 hours of initial presentation.

Initial electrocardiography and creatine kinase measurement gave normal results in all 110 cases. The patients had no further symptoms after discharge, and the repeat electrocardiograms all showed no abnormality. Cardiac troponin T was detected in eight (7%) patients at follow up (table 1). In seven the cardiac troponin T concentration was ≥ 0.1 μg/l, the level which indicates myocardial infarction. All patients with raised cardiac troponin T concentrations were subsequently referred for cardiac assessment and enrolled in the cardiac secondary prevention programme.

Discussion

We found missed myocardial damage of prognostic importance in 6% of patients sent home from the emergency department. The ability of raised cardiac troponin T concentration to predict risk of subsequent cardiac events has been well documented.[8 9] The size of the risk depends on how high the troponin T concentration is,[10 11] but in patients without electrocardiographic changes a cut-off of 0.1 μg/l is the optimal predictor of death. Some of the seven patients may have been considered to have unstable angina rather than non-Q wave myocardial infarction by conventional criteria. However, four patients had cardiac troponin concentrations above 0.5 μg/l, which has a 95% specificity for non-Q wave myocardial infarction. The risk of subsequent cardiac events in non-Q wave acute myocardial infarction is the same as that seen in patients with a cardiac troponin concentration above 0.1 μg/l.

Rates of missed acute myocardial infarction have usually been estimated by detailed review of case notes or by using follow up with a questionnaire or interview.[12–14] The measurement of cardiac troponin T to detect cardiac damage is a valuable addition to the range of tests for audit and quality assurance or for follow up clinics.

The incidence of missed acute myocardial infarction was estimated at 11.8% in a detailed audit of patients discharged from the emergency department,[12] although the figure usually quoted is 6-8%.[13 14] We studied 69% (110/160) of the total eligible population, and our audit is unlikely to have seriously underestimated or overestimated the number of patients missed (6%). Data frequently presented for the United States show that of 6.0 million patients attending the emergency department each year with chest pain, 5.7 million have non-diagnostic electrocardiograms; 4.1 million are sent home, of whom 75 000 (0.18%) have undiagnosed acute myocardial infarction. However, a recent study which measured cardiac troponin T and cardiac troponin I showed an incidence of prognostically important myocardial damage of 6% in patients without a diagnostic electrocardiogram on presentation.[15]

Patients with missed myocardial infarction or high risk unstable angina who are sent home have a high risk of subsequent cardiac events. Medical litigation for missed acute coronary syndromes in the United States accounts for 21-22% of malpractice claims,[16] with an estimated total cost of $1.8bn-$15bn (£1bn-£9bn) annually.[17] Medical litigation is also rising in the United Kingdom. More importantly, however, these patients are deprived of the opportunity to enter cardiac secondary prevention programmes, which will substantially improve their subsequent survival.

We have shown that measurement of cardiac troponin T can be used to assess the incidence of prognostically important myocardial damage in patients discharged from the emergency department. The test can be used to determine the effectiveness of other interventions to reduce misdiagnosis of chest pain, such as computer aided decision making protocols or serial biochemical testing.[18–24] In addition, measurement of cardiac troponin T at follow up is an easy and convenient method of risk assessment.

What is already known on this topic

Diagnosis is difficult in patients presenting with chest pain of unknown cause

Measurement of cardiac troponin T can reliably detect heart damage within 1-2 days after infarction

What this study adds

6% of patients discharged from the emergency department had troponin T concentrations suggesting important cardiac damage on review after 12-48 hours

Repeat electrocardiography on these patients showed no abnormality

Measurement of cardiac troponin T is a convenient diagnostic and audit tool for monitoring performance in emergency departments

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Commentary: Time for improved diagnosis and management of patients presenting with acute chest pain

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Cardiac troponins T and I are highly sensitive and specific markers for myocardial damage. Troponins are not detectable in normal serum, and modern immunoassays make diagnosis of minor degrees of cardiac damage possible. Cardiac troponins are detectable in all patients who have had a myocardial infarction within 12 hours of the onset of symptoms. They are also detectable in around 30% of patients with unstable angina. Those with higher troponin concentrations are at increased risk of a cardiac event over the ensuing weeks, and measurement of troponins can be used to guide acute treatment.[1 2] The conclusion of Collinson et al that detection of troponin T in patients discharged from the emergency department reflects missed diagnosis of acute coronary syndromes must be correct. The paper provides further evidence that our management of patients presenting with acute chest pain is far from perfect.

Most people presenting to emergency departments have medical conditions, and chest pain is by far the commonest. It is surprising, therefore, how few systematic data are available. Collinson et al's study is a welcome addition to the literature, but some aspects should be interpreted with caution. The 6% rate of missed myocardial infarction refers to a subgroup of patients in the study and not to the total number of patients presenting with suspected or confirmed myocardial infarction. Our study suggested that the rate of missed infarction was much lower.[3] The concentrations of troponin T reported certainly reflect myocardial damage but may not indicate acute myocardial infarction by currently agreed criteria. It is not clear whether the patients were divided prospectively into the four groups. There was no follow up of patients thought to have a non-cardiac problem, yet accurate diagnosis of these patients is notoriously difficult—for example, not all patients whose pain improves after antacid have gastrointestinal disease. Also, a third of the patients in the high risk group who were discharged were not followed up. The long half life of circulating troponin T means that the marker may have been positive at presentation in some of the patients, and it is a pity that it was not measured at presentation. The electrocardiogram appears abnormal in a large proportion of patients with unstable coronary syndromes but was not found to have prognostic value in this study. It is not clear whether the electrocardiograms were independently reviewed, and it is surprising that none of eight patients with supposed missed infarction developed abnormal electrocardiographic traces.

How, then, can we improve chest pain management? Even though markers of cardiac damage cannot

accurately diagnose myocardial damage in the first few hours after presentation,[4] logical combination of biochemical, clinical, and electrocardiographic data may improve early diagnosis.[5] The clinical situation is evolving, and it may be justified to admit patients to a low dependency observation area for serial electrocardiography and biochemical tests.[6 7] Accurate diagnosis could be made within 12 hours in most cases. Finally, urgent follow up of all discharged patients in whom cardiac disease was not fully excluded could be justified as a routine. Measurement of troponin T or I, along with repeat electrocardiography, would certainly help to ensure that high risk patients were not missed.

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Infant mortality, stomach cancer, stroke, and coronary heart disease: ecological analysis

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Mortality from stomach cancer and stroke shows an international correlation, consistent inverse socioeconomic gradients, a particular dependence on socioeconomic circumstances in childhood,[1] and parallel patterns of decline in most industrialised countries over the past 30-40 years. The plausibility of the hypothesis that salt intake underlies this similarity has been weakened over the past decade as evidence for *Helicobacter pylori* as the key factor in the aetiology of non-cardia stomach cancer has increased.[2] *H pylori* is thought to be acquired in childhood, and risk of infection is closely related to living conditions, hygiene, and housing standards. Geographical, socioeconomic, and secular variations in the prevalence of *H pylori* fit well with the corresponding trends and differences in mortality from stomach cancer between and within countries.[2]

Infant mortality in the early part of the 20th century indicates living conditions and, in particular, standards of hygiene. We investigated how far international variations in infant mortality in the past predict adult mortality today from stomach cancer, stroke, and other causes.

Subjects, methods, and results

Death rates from stomach cancer and other causes were obtained from a database of the World Health Organization (www.who.int/whosis/mort/download.htm). We calculated sex specific mortality in

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Relation of adult mortality (age 65-74 years in 1991-3) with infant mortality at time of birth and at time of death for 27 countries*

	Infant mortality 1921-3				Infant mortality 1991-3			
	Male		Female		Male		Female	
	Coefficient	P value	Coefficient	P value	Coefficient	P value	Coefficient	P value
Pearson correlation coefficients:								
All causes	0.52	0.005	0.51	0.007	0.58	0.002	0.63	<0.001
Respiratory tuberculosis	0.77	<0.001	0.73	<0.001	0.40	0.04	0.33	0.09
Stomach cancer	0.83	<0.001	0.82	<0.001	0.39	0.04	0.44	0.02
Lung cancer	–0.10	0.61	–0.48	0.01	–0.02	0.91	–0.23	0.24
Coronary heart disease	–0.05	0.81	0.16	0.42	0.13	0.53	0.28	0.16
Stroke	0.66	<0.001	0.63	<0.001	0.61	<0.001	0.64	<0.001
Partial correlation coefficients†:								
All causes	0.32	0.11	0.28	0.17	0.42	0.03	0.50	0.009
Respiratory tuberculosis	0.71	<0.001	0.69	<0.001	0.01	0.96	–0.07	0.72
Stomach cancer	0.80	<0.001	0.77	<0.001	–0.08	0.71	0.04	0.87
Lung cancer	–0.10	0.60	–0.43	0.03	0.04	0.86	0.02	0.92
Coronary heart disease	–0.13	0.52	0.03	0.90	0.18	0.39	0.23	0.27
Stroke	0.51	0.008	0.45	0.02	0.42	0.03	0.48	0.01

*Australia, Austria, Belgium, Bulgaria, Canada, Chile, Czechoslovakia, Denmark, Finland, France, Greece, Hungary, Ireland, Italy, Japan, Netherlands, New Zealand, Norway, Poland, Portugal, Romania, Russian Federation, Spain, Sweden, Switzerland, United Kingdom, United States.
†Sex and cause specific correlations of adult mortality with infant mortality in one period adjusted for infant mortality in the other period.