ORIGINAL ARTICLE

ROMEO: a rapid rule out strategy for low risk chest pain. Does it work in a UK emergency department?

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Aims: To examine the feasibility of using the ROMEO (rule out myocardial events on “obs” ward) pathway for low risk patients with chest pain in a UK emergency department.

Methods: A prospective study was undertaken to determine outcomes for the first 100 patients entering the pathway (from May to Oct 1999). Serum troponin levels, serial ECG recordings, exercise test result, total length of stay, and final diagnoses were reviewed. Patients were telephoned after discharge to inquire about persisting or recurrent pain, and further investigations after completing the ROMEO pathway.

Results: 82 of 100 (82%) had myocardial damage excluded by serum troponin assay. Sixty two of 82 (76%) of these completed exercise tolerance testing (ETT). Fifty seven of 62 (92%) ETTs were negative. Twenty of 82 (26%) did not undergo ETT because of mobility problems, recent ETT, or if considered very low probability of cardiac pain on consultant review. Five of 100 (5%) had an increased initial troponin and five of 100 (5%) had an increased 12 hour troponin. These patients were referred for admission under the general physicians. Seven of 100 (7%) were referred for other reasons (late ECG changes, continuing or worsening pain). One patient self discharged. Length of stay varied because of changes to arrangements for ETT. The median time for all patients over the period studied was 23 hours. All patients were discharged within an hour of a negative ETT.

Follow up results: 67 of 74 (91%) eligible patients were contacted by telephone. Forty six of 67 (69%) had no further pain, attendances, or GP consultations. Six of 67 (9%) had further cardiological investigation or treatment.

Conclusions: A rapid rule out strategy such as the ROMEO pathway is feasible in the UK healthcare setting and provides standardised and consistent evaluation.

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The traditional UK approach to the management of patients with potentially cardiac chest pain but a normal ECG has been to admit formally to hospital under the general physician “take” process for serial cardiac enzymes and ECGs, with inpatient exercise testing for selected patients. Various centres in North America have shown the effectiveness of “rapid rule out” strategies for selected patients presenting to emergency departments with acute chest pain but very few UK emergency departments have so far instituted similar protocols.^{1,2}

Our objective was to examine the feasibility of a chest pain protocol in a UK general hospital emergency department and to review the outcomes of patients entered on the pathway. We present the data for the first 100 patients.

METHODS

The emergency department (ED) serves as the single portal of entry for all emergency referrals and admissions to the hospital, regardless of referral, with a catchment area of 480 000. All patients presenting with non-traumatic chest pain are immediately assessed in a high dependency area by a nurse who records an ECG. This is shown at once to a senior ED doctor as part of the triage process.

From 1 May 1999 all patients who self presented to the ED and fulfilled the criteria in box 1 were managed according to the ROMEO pathway.

Protocol

After initial ECG, assessment, and an urgent troponin I (TnI) and CK measurement, patients were admitted for standard cardiac monitoring in the emergency department observation unit. This is a 10 bed ward staffed by three nurses with 24 hour

cover by senior ED doctors. ECGs were repeated before transfer to the observation unit and 12 hours after pain onset—that is, a minimum of three ECGs. Additional ECGs were recorded if the patient had any further chest pain.

All other investigations such as haematology or chest radiography were ordered at the discretion of the admitting doctor.

A further blood sample for Tn I assay was taken 12 hours after the onset of chest pain. If this was between 11 00 pm and 8 00 am the blood was stored for laboratory assay at 8 00 am. During our study, all troponin assays performed were recorded on the biochemistry department database to facilitate subsequent audit.

On admission to the observation ward, eligible patients were booked for pre-discharge exercise tolerance testing (ETT) to be carried out on the confirmation of a negative 12 hour TnI assay. This was a standard Bruce protocol test administered by cardiac centre technicians, at the RUH Cardiac Centre on

Box 1 Inclusion criteria

- Age >25
- Acute, potentially cardiac, chest pain (central/left sided chest pain lasting more than 20 minutes)
- Physiologically normal—normotensive, P<100, not clammy, vomiting, or dyspnoeic at rest
- ECG normal/known longstanding changes (including LBBB/AF rate controlled)
- No myocardial infarction within past 12 months
- No suspicion of aortic dissection
- No other reason to prevent discharge on completion of the pathway
- ED observation ward bed available

Name	Follow up date
Admission date	
Hospital number	
Further episodes of chest pain?	Yes/No
If yes	
Seen GP?	Yes/No
Treatment?	
Working diagnosis?	
Further investigations?	
Further admissions?	
All patients	
Were you given a diagnosis?	

Figure 1 Follow up questionnaire.

weekdays. Patients who were admitted on Friday or Saturday were allowed to go home after a negative 12 hour TnI assay to await their ETT on Monday. They were advised to return to the ED immediately if they suffered any further chest pain.

Before discharge, all patients were reviewed by a senior ED doctor to discuss their results, and were given a GP letter detailing the pathway and the patient's results. A working diagnosis of the cause of chest pain was given where appropriate. In other cases, it was explained to patients that a cause had not been found. All patients were advised to seek follow up with their GP if any pain recurred.

Criteria for leaving pathway

- (1) TnI concentrations greater than 1.0 µg/l (later changed to 0.3 µg/l—see discussion)
- (2) ECG changes during admission consistent with acute ischaemia (that is, ST segment or T wave changes, new arrhythmia or conduction delay).
- (3) Any signs or symptoms suggesting an unstable medical condition requiring admission for further investigation or treatment.
- (4) Patients who had a positive ETT were seen immediately by a senior member of the cardiology team for assessment and further management.

ECG review

At the end of the data collection period of this study, we sought to confirm that patients had not been inappropriately admitted to the ROMEO pathway with missed signs of ischaemia on their ECG. A consultant cardiologist who was blinded to any other detail, retrospectively reviewed 25 of 26 initial ECGs of patients who did not complete the ROMEO pathway (one patient's ECG was lost). None of these ECGs was considered to show changes that would have made the patient ineligible to enter the ROMEO pathway.

Follow up

Follow up with a standard telephone questionnaire was carried out by two authors (CT and AF-H) in February 2000. The number of calendar months from admission until follow up ranged from 4 to 10 months. The mean was 6.51 months and the median period was seven months.

We attempted to contact all patients who had fulfilled the "rule out" criteria and been discharged from the emergency observation ward without other follow up arranged (that is, those with a negative 12 hour troponin assay and completely normal, or omitted, ETT).

No further attendances by those patients lost to follow up were found on a search of RUH hospital records.

Outcome measures

On completion of the pathway, a negative 12 hour TnI was considered to rule out myocardial damage³ and identify those

at low risk of coronary events,³⁻⁶ and the addition of a normal ETT was taken to indicate an acceptably low probability of chest pain attributable to simple angina. A normal ETT was defined as one with no ST changes. Failure to complete the 12 minute protocol, or chest pain without ST change was reported but not considered to be a positive test.

Secondary outcome measured was the length of time to completion of the pathway.

Follow up outcomes measured were adverse events and further cardiology investigation.

RESULTS

Seventy two patients were male. Mean age was 49 years (range 25–80, median 56). Twenty eight patients were female. Mean age was 55 years (range 32–85, median 55).

Five patients were found to have an initial troponin level of >1.0 µg/l

- Four of these patients were subsequently confirmed on ECG or angiography to have an acute coronary syndrome.
- One patient was investigated for arrhythmia related angina.

Eight patients left the ROMEO pathway before the 12 hour troponin level was measured

- One had persistent severe chest pain. Subsequent serial creatine kinase measurement was normal and was followed by a normal ETT. The patient was discharged to have a thallium scan as an inpatient (in Spain).
- One patient with an initial troponin of 0.5 µg/l (see Discussion) had persistent chest pain. myocardial infarction was diagnosed on serial cardiac enzymes.
- One patient with a marginally increased creatine kinase (maximum 216) on admission complained of persistent pain. Subsequent ETT was normal. The patient declined angiography and self discharged.
- Three patients developed ECG changes on the observation ward before the 12 hour troponin assay. One of these had an initial troponin concentration of 0.7 µg/l (see Discussion).
- One patient was referred with persistent chest pain. Subsequent serial enzymes and ETT were normal.
- One self discharged and refused follow up.

Five patients had an increased troponin I at 12 hours. All were found to have acute coronary syndromes

- One (TnI=1.1 µg/l) had a subsequent positive ETT.
- One (TnI=8.0 µg/l) subsequently developed ST elevation on ECG and a raised creatine kinase (249). Diagnosis: inferior subendocardial infarct.
- One (TnI=18.3 µg/l) had subsequent angiography and coronary artery bypass graft.
- One (TnI=1.8 µg/l) had positive exercise test and went on to angioplasty.
- One (TnI=109.8 µg/l). Diagnosis of myocardial infarction confirmed on serial cardiac enzyme testing.

Five patients had a positive ETT

All were reviewed immediately by cardiology registrar, staff grade, or consultant.

- Four were discharged on medication for early outpatient angiography.
- One had a strongly positive ETT and was admitted to CCU pending urgent angiography.

Twenty patients did not have ETT

- Thirteen of these were reviewed by an ED consultant after negative 12 hour troponin assay and considered to have a clearly non cardiac cause of chest pain.

Table 1 Follow up results

	ETT group 49 (73)	No ETT group 18 (27)	Total respondents 67
No further chest pain	35 (71)	11 (60)	46 (69)
Further chest pain	14 (29)	7 (39)	21 (31)
Subsequent GP visit for chest pain	13 (27)	5 (28)	18 (27)
Further cardiology referral	2 (4)	5 (28)	7 (10)
Reattended ED with chest pain	1 (2)	0	1 (1.5)

Percentages are shown in parentheses.

- Six were physically unable to perform ETT.
- One had an ETT already booked by her GP.

Three patients had a normal ETT but complained of severe pain on exercise

- One was subsequently discovered to have known Münchhausen's syndrome
- One was referred for an urgent neurology opinion because of arm pain on exercise with subsequent diagnosis of cervical root syndrome.
- One was admitted for further investigation and found to have a mesothelioma

Length of time to completion of ETT

For patients who were admitted from Sunday to Thursday the mean time to completion of ETT was 26.4 hours. Patients who were admitted on Friday or Saturday were allowed home after a negative 12 hour troponin to return on Monday for their ETT.

The median time to ETT for all patients was 23 hours with a range of 2.25 to 124 hours. The shortest time to ETT was in a patient who presented over 12 hours after the onset of chest pain and therefore did not have any delay to the 12 hour troponin measurement. Almost all patients were discharged within an hour of a negative ETT.

Follow up results

The final response rate was 67/74 (90.6%) of eligible patients. Forty nine of 67 (73.13%) patients contacted had had an ETT (table 1). Seven of 67 (10%) patients had a cardiology referral organised by the GP. One of these patients was subsequently found to have paroxysmal atrial fibrillation causing angina. No cardiac cause for chest pain was identified in the other six.

Of the 74 patients discharged from the ROMEO Pathway, only one patient (from the ETT group) re-attended the accident and emergency department with chest pain. She was referred as an outpatient to a cardiologist and found to have no cardiac cause for pain.

Other investigation

Seven of 67 (10%) were referred for endoscopy (all from the group who had undergone ETTs). Six of these had positive findings.

Summary of key findings (see fig 2)

We were able to rule out myocardial damage in 82% of patients on serum TnI assay. Sixty two patients completed exercise tolerance testing and 57 of these exercise tests were negative.

Box 2 Diagnoses given for cause of chest pain (on discharge or subsequently by GP)

Non-specific chest pain 21 (31%)
 Gastrointestinal 20 (30%)
 Musculoskeletal 8 (12%)
 Psychosomatic 8 (12%)
 Other non-cardiac 6 (9%)
 Paroxysmal atrial fibrillation 1 (1.5%)

Figure 2 Summary of results.

Thus, of 100 patients entered on the ROMEO pathway, 77% of patients were discharged, 20% after rule out of myocardial damage alone, and 57% with a diagnosis of non-cardiac chest pain. Of those 22 patients referred for further cardiology investigation from the pathway, 18 were confirmed to have cardiac chest pain, 13 of whom had acute coronary syndrome.

The mean time to completion of the pathway on weekdays was 26.4 hours.

No adverse events were identified at follow up.

DISCUSSION

It has long been recognised that a small percentage of patients presenting to the ED with acute chest pain but normal ECG have ischaemic cardiac pain. In the United States, Fesmire⁷ found a 2% incidence of AMI in such patients in 1989; a 4% incidence of unstable angina pectoris in a similar group of patients was reported by Lee in 1985.⁸

A UK audit in 1989 found an 11.8% incidence of missed acute ischaemic heart disease.⁹ As a result, UK management has been to admit large numbers of patients to hospital for two to three days for serial ECGs and cardiac enzymes. Selected patients are then investigated further using exercise tolerance testing. However, despite changes in management strategies over the past decade, the most recent studies

indicate that ischaemia is still undiagnosed in emergency departments on both sides of the Atlantic in 2% to 6% of "low risk" patients.^{10 11}

The need to prevent inadvertent discharge of such patients, combined with the drive to reduce costs, has resulted in 22% of emergency departments in the US instituting "rule out" protocols in chest pain assessment units (CPAU).¹² The majority of published studies from these units describe pathways using CKMB assay to rule out acute myocardial infarction. The data relating to chest pain observation units have recently been comprehensively reviewed.^{1 2} We have not sought to compare the outcomes of patients on the ROMEO pathway directly with previously reported rule out pathways because all the literature comes from the USA and relates to a different patient population. Furthermore, none of these studies has used troponin as the cardiac marker.

A few UK emergency departments are introducing "rule out" pathways using CKMB or TnT combined with exercise tolerance testing,^{13 14} but as yet few data have been published. There is ongoing debate about the most appropriate cardiac markers but there is evidence that troponin assay has a stronger negative predictive value than CKMB mass, approaching 100% 8–12 hours after pain onset.³ Troponin assay also enables clearer risk stratification of patients with unstable angina.^{3–6} TnT is slightly less cardio-specific than TnI as it may be falsely positive in renal failure.

ETT is used in the ROMEO pathway to identify patients with simple angina pectoris and is safe in all patients in whom acute myocardial infarction and high risk unstable angina pectoris have been ruled out by serum troponins.¹⁵

Although it is recognised that a negative ETT does not have a 100% negative predictive value for angina pectoris,¹⁶ it allows further risk stratification and the identification of a group with an extremely low risk of coronary events in the next 12 months. Those patients with a low probability of ischaemic heart disease, but a high risk of incomplete exercise test were not put forward for the ETT.

In our study, patients had creatine kinase activities measured on entering the pathway as well as TnI. Those patients who were referred before their 12 hour troponin test went on to have standard serial enzyme testing but did not have further troponin levels measured. It was also considered necessary to have a recorded creatine kinase measurement as the World Health Organisation diagnostic criteria for myocardial infarction remain based on creatine kinase rise.

Learning points

The data presented here are for the first 100 patients managed with this pathway and various improvements were made during the study period.

We initially chose a TnI concentration of over 1.0 µg/l as the threshold for referral for admission. This cut off was advised by the RUH biochemistry department, based on information from the assay manufacturer. However, this threshold was lowered to 0.3 µg/l after confirmation of ischaemia in two patients with admission levels of 0.5 and 0.7 µg/l. No other patient in the study had TnI between 0.3 and 1.0 µg/l. A measurement of 0.3 µg/l is the lowest recordable level of TnI on the assay used. This is specific for the assay used and cannot be extrapolated to other units or studies.

It would be possible to use a qualitative troponin test instead of a quantitative test if risk stratification of those patients with unstable angina did not affect subsequent patient management. Near patient testing kits are available for qualitative testing, but we felt that there was a risk of inappropriate use and interpretation of these tests if they were easily available in the department.

Most of the patients managed on the pathway but not put forward for ETT after consultant review presented early in the study period. In September 1999 a full shift registrar/staff

grade rota was introduced, so that all patients entering the pathway were screened by experienced doctors. This led to a marked fall in such decisions.

The weak link in the ROMEO pathway was the delay awaiting ETT and we believe that our reliance on another department for this service is an inherent weakness. The Northern General Hospital in Sheffield has developed their own ED based ETT service as part of a "rule out" strategy¹³ and we aim to incorporate this into the ROMEO pathway.

Another possible shortcoming in our pathway was that it did not include alternative testing for the 6% of patients who were physically unable to undergo ETT. Both dobutamine stress echocardiography and thallium scanning have been used in similar rule out protocols in the US^{17 18} and we are considering extending our pathway similarly.

The American experience of CPOUs has shown that costs can be significantly reduced without compromising patient safety^{19–22} and a recent UK study has suggested potential for cost savings in one UK hospital.²³

We have not attempted to compare the ROMEO pathway prospectively with traditional inpatient management at our institution, but the results of this initial study support such a pathway as a suitable standard for comparison in a prospective randomised controlled trial in UK hospitals.

Given the success of our early experience with the ROMEO pathway, we hope that this paper provides some support to those planning to introduce similar pathways in other EDs.

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Contributors

Steve Meek initiated the ROMEO pathway at Bath and designed the protocol with assistance from William Hubbard and Andrew Taylor. Clare Taylor and Anna Forrest-Hay collected the data and carried out the follow up survey. Clare Taylor and Steve Meek wrote the paper and are its guarantors.

APPENDIX 1 SUMMARY OF DECISION MAKING CRITERIA

- 1 Patient fulfills entry criteria (see box 1): enter on ROMEO pathway
- 2 Raised TnI on admission: refer to on take physicians
- 3 If patient physically capable: ETT provisionally booked pending normal 12 hour TnI
- 4 Persistent, severe, chest pain or ECG changes suggestive of ischaemia: refer before 12 hour TnI
- 5 If TnI normal: ETT unless considered clearly non-cardiac pain on review by senior ED doctor
- 6 Positive ETT: immediate review and subsequent management by cardiologist
- 7 If non-positive ET: discharge by ED doctor with working diagnosis and letter to GP

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REFERENCES

- 1 Goodacre SW. Should we establish chest pain observation units in the UK? A systematic review and critical appraisal of the literature. *J Accid Emerg Med* 2000;17:1–6.

- 2 **Quinn G**. Chest pain evaluation units. *J Accid Emerg Med* 2000;**17**:237–240.
- 3 **Hamm CW**, Goldmann BU, Heeschen C, *et al*. Emergency room triage of patients with acute chest pain by means of rapid testing for cardiac troponin T or I. *N Engl J Med* 1997;**337**:1648–53.
- 4 **Hamm CW**, Ravkilde J, Gerhardt W, *et al*. The prognostic value of serum troponin T in unstable angina. *N Engl J Med* 1992;**327**:192–4.
- 5 **Ohman EM**, Armstrong PW, Christenson RH, *et al*. Cardiac troponin levels for risk stratification in acute myocardial ischaemia. *N Engl J Med* 1996;**335**:1333–41.
- 6 **Luscher MS**, Thygesen K, Ravkilde J, *et al*. (TRIM study group) Applicability of cardiac troponin T and I for early risk stratification in unstable coronary artery disease. *Circulation* 1997;**96**:2578–85.
- 7 **Fesmire FM**, Percy RF, Wears RL, *et al*. Initial ECG in Q wave and non-Q wave infarction. *Ann Emerg Med* 1989;**18**:741–6.
- 8 **Lee TH**, Cook F, Weisberg M, *et al*. Acute chest pain in the emergency room: identification and examination of low risk patients. *Arch Intern Med*. 1985;**45**:65–9.
- 9 **Emerson PA**, Russel NJ, Wyatt J, *et al*. An audit of doctors' management of patients with chest pain in the accident and emergency department. *Q J Med* 1989;**70**:213–20.
- 10 **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.
- 11 **Collinson PO**, Premachandram S, Hashemi K. Prospective audit of incidence of prognostically important myocardial damage in patients discharged from emergency department. *BMJ* 2000;**320**:1702–5.
- 12 **Zalenski JR**, Rydman RJ, Ting S, *et al*. A national survey of emergency department chest pain centers in the United States. *Am J Cardiol* 1998;**81**:1305–9.
- 13 **Goodacre S**, Morris FP, Angelini K, *et al*. A descriptive study of a chest pain observation unit in a UK hospital. [Abstract]. *J Accid Emerg Med* 2000;**17**:58.
- 14 **Richell Herren KR**, Mackway-Jones K, Richards CR, *et al*. Early recognition of myocardial damage: an emergency department based diagnostic test study. [Abstract]. *J Accid Emerg Med* 2000;**17**:59.
- 15 **Lindhal B**, Andren B, Ohlsson J, *et al*. (FRISK study group) Risk stratification in unstable coronary artery disease. Additive value of troponin T determinations and pre discharge exercise tests. *Eur Heart J* 1997;**18**:762–70.
- 16 **Polanczyk CA**, Johnson PA, Hartley LH, *et al*. Clinical correlates and prognostic significance of early negative exercise tolerance testing in patients with acute chest pain seen in the hospital emergency department. *Am J Cardiol* 1998;**81**:288–92.
- 17 **Mikhail MG**, Smith FA, Gray M, *et al*. Cost effectiveness of mandatory stress testing in chest pain center patients. *Ann Emerg Med* 1997;**29**:88–98.
- 18 **Farkouh ME**, Smars PA, Reeder GS, *et al*. A clinical trial of chest pain observation units for patients with unstable angina. *N Engl J Med* 1998;**339**:1882–8.
- 19 **Nichol G**, Walls R, Golman L, *et al*. A critical pathway for management of patients with acute chest pain who are at low risk for myocardial ischaemia: recommendations and potential impact. *Ann Intern Med* 1997;**127**:996–1005.
- 20 **Gaspaz JM**, Lee TH, Weinstein MC, *et al*. Cost effectiveness of a new short stay unit to 'rule out' myocardial infarction in low risk patients. *J Am Coll Cardiol* 1994;**24**:1249–59.
- 21 **Roberts RR**, Zalenski RJ, Mensah EK, *et al*. Costs of an emergency department based accelerated diagnostic protocols. Hospitalization in patients with chest pain. A randomized controlled trial. *JAMA* 1997;**278**:1670–6.
- 22 **Gomez MA**, Anderson JL, Karagounis LA, *et al*. An emergency department based protocol for rapidly ruling out myocardial ischaemia reduces hospital time and expense. Results of a randomized study. (ROMIO). *J Am Coll Cardiol* 1996;**28**:25–33.
- 23 **Goodacre S**, Morris F, Arnold J, *et al*. Is a chest pain observation unit likely to be a cost saving in a British hospital? *Emerg Med J* 2001;**18**:11–14.

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