ORIGINAL ARTICLE

Potential health promotion benefits of lipid testing for all patients presenting with chest pain to an emergency department

C V Elder, D Kerr, R X Davey, A-M Kelly

Emerg Med J 2006;**23**:23–26. doi: 10.1136/emj.2004.023184

See end of article for authors' affiliations

Correspondence to: Mrs D Kerr, Joseph Epstein Centre for Emergency Medicine Research; Western Hospital, Private Bag, Footscray 3011; Debbie.Kerr@wh.org.au

Accepted for publication 29 March 2005

Objectives: To establish the prevalence of previously undiagnosed dyslipidaemia in patients presenting to the emergency department (ED) with non-traumatic chest pain and, more particularly, the prevalence in the subgroup which was discharged home from the ED, the group that traditionally would not have received a lipid test.

Methods: Prospective, observational study of adult patients presenting to an ED with non-traumatic chest pain as the presenting complaint.

Results: A total of 185 eligible patients underwent lipid testing during their presentation: 96 in the ED and 89 in the wards. Overall 61% (n = 112) of patients had at least one abnormal lipid level. Of patients discharged from the ED, 62% had at least one abnormal lipid level.

Conclusions: A moderate, but useful, increase in detection rates of dyslipidaemia is possible if lipid testing is offered to all patients presenting with chest pain, and not just to those who are admitted to wards for further investigation and management of suspected acute coronary syndromes. Testing of this group should be considered as a health promotion initiative in the ED, with appropriate follow up in the community.

Coronary heart disease (CHD) is the single leading cause of death in Australia, and it accounted for around 21% of deaths in 2000.[1] It is estimated that CHD will be the world's leading public health problem by 2020.[1] Risk factors for developing CHD include a family history of CHD, age greater than 45 years, obesity, dyslipidaemia, diabetes, chronic renal failure, hypertension, and smoking.[2] Some of these risk factors are modifiable whereas others are not. With respect to dyslipidaemia, it is estimated that if the Australian national mean cholesterol level were reduced by 0.5 mmol/l, coronary events for the population would reduce by 13% per year.[3] Methods to achieve this might include exercise, drug therapy, and dietary control.

It has been suggested that screening for dyslipidaemia should be focused on patients already at increased risk of CHD, for example hypertensive patients.[2] Some limited research has investigated the usefulness of cholesterol screening of selected patient groups in the emergency department (ED) as a primary prevention strategy and found unexpectedly high levels of dyslipidaemia.[4–6] In Australia, the National Heart Foundation (NHF) advocates testing the fasting lipid levels in all patients who present to hospital with unstable angina within 24 hours of onset of symptoms.[2] Currently, most patients who present with non-traumatic chest pain to Australasian EDs only receive lipid screening if they are admitted for further investigation of suspected CHD.

The aims of this study were to establish the overall prevalence of previously undiagnosed dyslipidaemia in patients presenting to the ED with non-traumatic chest pain and, more specifically, the prevalence in the subgroup that is discharged home from the ED, the group that traditionally would not receive a lipid test.

METHODS

Setting

We undertook this study at Western Hospital, Footscray, Victoria, Australia, a tertiary care community teaching hospital with an annual ED census of 34 000 patients, of whom approximately 1800 present with chest pain.

Design

Prospective, observational study, with data collected from medical records and pathology databases during a five month period (October 2003 – February 2004).

Study protocol

We asked ED medical and nursing staff to take blood for lipid studies from all adult patients presenting with non-traumatic chest pain. Compliance with the new protocol was encouraged via educational sessions for both medical and nursing staff before and during the study period, prominent display of reminder posters, easy access to prepared pathology request slips, and personal reminders by researchers on an ad hoc basis.

All patients identified from the ED data management system (HASS) with an ED discharge diagnosis of chest pain, acute myocardial infarction, angina pectoris, and coronary artery disease for the study period were considered for inclusion into the study. We excluded patients aged less than 16 years, taking medication for dyslipidaemia or presenting with trauma related chest pain. For patients with multiple ED presentations with chest pain over the study period, only the first visit was included.

We collected data on patient demographics (age, sex, country of birth), presenting complaint, if the patient was already prescribed lipid altering medication, results of lipid measurements, presence of CHD risk factors, ED discharge destination, ED final diagnosis, and for those who were hospitalised, the final hospital diagnosis.

Abbreviations: Apo B, apolipoprotein B; CHD, coronary heart disease; ED, emergency department; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; NHF, National Heart Foundation; TC, total cholesterol

One researcher (CE) collected the data. For assessing the interrater reliability for three variables (previous lipid lowering medication, high total cholesterol (TC), and presenting complaint of chest pain) an independent researcher experienced in reviewing medical files (DK) reviewed 10% of the sample. There was excellent agreement for all three data extractions: previous lipid lowering medication, $\kappa = 0.91$; high TC result, $\kappa = 1.00$; and presenting complaint of chest pain, $\kappa = 1.00$.

Outcome measures

The primary outcome measures were the prevalence of dyslipidaemia in this patient population and the increase in detection of dyslipidaemia that resulted from testing lipids in the ED. Dyslipidaemia was defined as any value exceeding the NHF[2] targets. For the purposes of this study, abnormal lipid levels were based on the target lipid levels defined by the NHF[2]: TC >5.0 mmol/l; triglycerides >2.0 mmol/l; high density lipoprotein cholesterol (HDL-C) <0.8 mmol/l; low density lipoprotein cholesterol (LDL-C) >3.0 mmol/l; and apolipoprotein B (Apo B) >1.0 mmol/l.

Samples were taken from patients in fasted and non-fasted states. As the calculated LDL-C is only considered an accurate measurement when the accompanying triglycerides is <4.5 mmol/l, usually assured by an 8–10 hour fast,[7] Apo B levels were explored as a substitute measurement for LDL-C. LDLs are always associated with the lipoprotein Apo B in a 1:1 ratio.[8] Serum TC, Apo B, and HDL-C levels can be tested, moreover, in either the fasted or non-fasted state.[9 10] Apo B was assayed on a Beckman Array analyser (St Vincent's Hospital, Melbourne), TC and triglycerides on a Beckman Synchron CX7 analyser (Western Hospital) and HDL-C on a Beckman Synchron CX5 analyser (Western Hospital) (Beckman Instruments Inc., Brea, CA).

Data analysis

Data were collected and entered into a Microsoft Access database for storage and analysis. We analysed the data with Analyze-IT software using descriptive statistics, χ^2 analysis, and odds ratio (OR) with 95% confidence intervals. Kappa agreement was used to describe interrater reliability.

Patients found to have abnormal lipids in the course of the study were notified of their results in writing and advised to seek information about treatment from their local doctor. This process was not specific to this study but is a standard process used by the Western Hospital ED to notify patients of abnormal results that require follow up. We did not follow up patients if they sought advice from their local practitioner about their lipid status.

The Melbourne Health Research Directorate Human Research Ethics Committee approved the study.

RESULTS

During the study period there were 754 eligible ED presentations by 660 patients, of which 379 presentations were excluded from data analysis for reasons shown in fig 1. A total of 185 patients underwent lipid testing and 190 did not. Patient demographics, ED diagnosis, disposition, past CHD history, CHD risk factors, and previous lipid testing data of these two groups of patients are shown in table 1. Male patients were more likely to have been tested for lipids (OR 1.7, p = 0.014). Among CHD risk factors, only a family history of CHD was more likely to induce testing (OR 2.3, p = 0.001). The chance of being tested increased steadily from undifferentiated chest pain (OR 0.3) through to that caused by an acute coronary event (OR 3.9, p<0.0001), with patients admitted to the coronary care unit (CCU) most likely to be tested.

Previous lipid studies had been normal in 27% patients and 20% had previously been diagnosed with dyslipidaemia. The lipid status of the remaining 53% of patients was not known. Of the 375 patients, 185 (49%) underwent lipid testing during their presentation (table 2): 96 in the ED and 89 in the wards. Overall 61% of patients (n = 112) had at least one abnormal lipid level (TC, LDL-C, or Apo B). Of patients discharged from the ED, a similar proportion (62%) had at least one abnormal lipid level. Rates of abnormality for the lipid markers in the discharged group were TC 60% (95% CI 45% to 73%), Apo B 54% (95% CI 39% to 68%), LDL-C 41% (95% CI 26% to 58%). Of patients discharged from the ED, 31% (14/42) had abnormal levels for all three lipid measurements (TC, Apo B, and LDL-C).

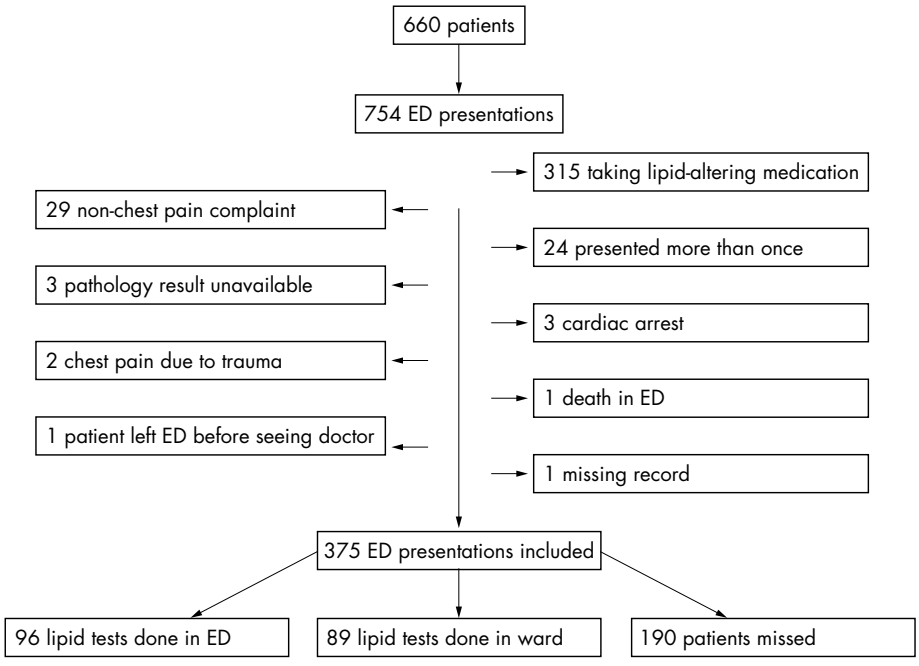

Figure 1 Sample derivation.

Table 1 Patient characteristics and outcomes for tested and non-tested groups*

	All patients	Test subgroups		OR (95% CI); p value
		Test not done	Test done	
No of patients	375	190	185	
Age in years (mean (range))	61.4 (21–96)	63.0 (21–96)	59.7 (21–91)	p=0.049
Sex (male)	204 (54)	91 (48)	113 (61)	1.7 (1.1 to 2.6); 0.014
ED diagnostic category				
Chest pain	231 (62)	145 (76)	86 (46)	0.3 (0.2 to 0.4); <0.0001
Angina	76 (20)	28 (15)	48 (26)	2.0 (1.2 to 3.4); 0.010
Myocardial infarction	68 (18)	17 (9)	51 (28)	3.9 (2.1 to 7.0); <0.0001
Disposition				
Coronary care unit	154 (41)	34 (18)	120 (65)	8.5 (5.3 to 13.7); <0.0001
Other ward	59 (16)	36 (19)	23 (12)	0.6 (0.3 to 1.1); 0.111
Home	162 (43)	120 (63)	42 (23)	0.2 (0.1 to 0.3); <0.0001
Risk factors				
CHD family history	95 (25)	34 (18)	61 (33)	2.3 (1.4 to 3.7); 0.001
CHD personal history	132 (35)	74 (39)	58 (31)	0.7 (0.5 to 1.1); 0.152
Renal disease	20 (5)	10 (5)	10 (5)	1.0 (0.4 to 2.5); 1.0
Obesity	49 (13)	21 (11)	28 (15)	1.4 (0.8 to 2.6); 0.308
Hypertension	172 (46)	89 (47)	83 (45)	0.9 (0.6 to 1.4); 0.779
Current smoker	95 (25)	44 (23)	51 (28)	1.3 (0.8 to 2.0); 0.388
Ex-smoker	82 (22)	36 (19)	46 (25)	1.4 (0.9 to 2.3); 0.207
Diabetes mellitus	70 (19)	35 (18)	35 (19)	1.0 (0.6 to 1.7); 1.0
Previous lipid testing				
Known abnormal	75 (20)	39 (21)	36 (20)	0.9 (0.6 to 1.6); 0.898
Known normal	100 (27)	42 (22)	58 (31)	0.6 (0.4 to 1.0); 0.056
Unknown	200 (53)	109 (57)	91 (49)	1.4 (0.9 to 2.1); 0.138

*Except for age all values are n (%).
CHD, coronary heart disease.

DISCUSSION

We found that a high proportion of patients investigated in the ED for chest pain have abnormal lipids. A moderate, but useful, increase in detection rates of dyslipidaemia was observed when lipid testing was offered to all patients presenting with chest pain and not just to those who were admitted to wards for further investigations and management of a suspected acute coronary syndrome.

Of the initial 754 ED presentations considered for inclusion into the study, 315 (42%) were excluded because the patients were already receiving lipid altering medication. This is a substantial proportion, and it suggests that many people at risk of CHD in Australia are undergoing lipid screening in the community with initiation of treatment if dyslipidaemia is detected.

Our findings are similar to those of two other recent studies, both conducted in America. Both found unexpectedly elevated rates of dyslipidaemia in ED patients presenting with chest pain: 50% with dyslipidaemia and 25% with high TC on capillary testing.[5 6] The patients discharged home from the Western Hospital ED, who would traditionally have not had lipid screening during their hospital visit, also showed a high rate of dyslipidaemia: 60% for elevated TC alone, and 62% for one of TC, LDL-C, or Apo B being elevated. Different thresholds for abnormality were used in the three studies, however, which makes further comparison difficult.

Detection raises the opportunity for intervention to correct dyslipidaemia and hopefully prevent the associated complications. Screening of lipids in this group of patients would appear to carry some potential benefit in terms of preventive action and should be considered as a health promotion initiative in the ED. All patients discharged from the ED who had abnormal lipids were notified by mail of their results and consultation with their doctor recommended. We are unable to report what proportion acted on this notification. Failure to act on the results would clearly undermine any potential benefit of detection.

Although it was our aim to test all patients with suspected acute coronary syndromes, patients were more likely to have undergone lipid tests if they were younger, male, were admitted to the CCU, had a more severe final diagnosis or had lipid results previously known to be normal. Except for family history of CHD, however, presence or absence of CHD risk factors did not affect test ordering. None of this is consistent with the recommendation of the Australian NHF, which advises that screening be offered to all patients with risk factors for CHD. We are unable to comment on why this may have occurred as this study was not designed to explore this issue.

Table 2 Dyslipidaemia detection rates amongst tested patients

Assay (abnormal)	All patients tested			Admitted patients			Discharged patients			Percentage increase in dyslipidaemia detection, p value
	n	Abnormal (%)	95% CI	n	Abnormal (%)	95% CI	n	Abnormal (%)	95% CI	
TC (>5.0 mmol/l)	185	105 (57)	50 to 64	143	80 (56)	48 to 64	42	25 (60)	45 to 73	14%, p 0.013
LDL-C (>3.0 mmol/l)	133	69 (52)	44 to 60	99	55 (56)	46 to 66	34	14 (41)	26 to 58	11%, p 0.110
Apo B (>1.0 mmol/l)	77	52 (68)	57 to 77	38	31 (82)	70 to 94	39	21 (54)	39 to 68	27%, p 0.001
TC or LDL-C, or Apo B	185	112 (61)	53 to 67	143	86 (60)	52 to 67	42	26 (62)	47 to 75	14%, p 0.009
HDL-C (<0.8 mmol/l)	145	16 (11)	7 to 17	105	12 (11)	5 to 17	40	4 (10)	4 to 23	3%, p 0.552
Triglycerides (>2.0 mmol/l)	184	55 (30)	24 to 37	142	39 (27)	20 to 34	42	16 (38)	25 to 53	9%, p 0.073

The use of Apo B in this study, although unusual, succeeded in its aim. It enabled the finding of dyslipidaemic patients in the ED whose total lipid picture otherwise would have remained unclear because they were not fasting. Among the 77 patients whose Apo B was determined, 68% had elevated levels, making Apo B the most sensitive of the dyslipidaemia markers used. Among the patients discharged from the ED, 21/39 (54%) had elevated Apo B levels compared with only 41% (14/34) with elevated LDL-C. The Apo B assay is now simple, inexpensive, and reliable. Results can be available from the laboratory before patients are discharged. Its use could beneficially change the way we go about CHD case finding, particularly in environments like the ED where immediate results, information, and education could powerfully reinforce the message to the patient.

The significant levels of dyslipidaemia identified in this study raise the question of whether this is isolated to the chest pain subgroup of ED patients or is only a reflection of high levels of dyslipidaemia in the particular local population. Dyslipidaemia is a recognised risk factor for coronary artery disease and, as noted above, has been found to be more prevalent in patients presenting with chest pain to EDs in America.[5 6] We chose to concentrate on this population of expectedly at-risk patients and did not include a control group with no chest pain but the broader question can be approached from Australia-wide data. The most recent available data comes from the Australian Diabetes, Obesity and Lifestyle Study 1999–2000 risk factor prevalence survey.[11] It found that among 6 406 465 Australians aged over 25 years 51.5% had a total cholesterol ⩾5.5 mmol/l. This level is 0.5 mmol/l higher than the threshold we used, and can only be taken as indicative of risk levels in the population presenting to our hospital, but nonetheless suggests that our 61% overall abnormality found is probably different from the local population's level.

This remains an area for future research, as it would help define the scope of any proposed screening/health promotion project in EDs. Current Australian guidelines for preventive activities in general practice recommend screening for people without risk factors from the age of 45 years.[12]

The role of ED in health screening and health promotion is controversial. Although both tasks are important, the appropriateness of the ED setting for these activities can be questioned. Patient numbers, illness severity, and follow up issues are all competing priorities. On the other hand, an ED workup for chest pain is an ideal time to reinforce strategies to prevent and mitigate evolving CHD. Making it work would rely on effective strategies to identify the abnormal results and follow up the affected patient.

Limitations of the study

This study has several limitations. Firstly, a large number of patients who were eligible for ED screening did not receive a lipid test. Workload and time pressures on staff may have contributed to the low number of enrolments. Staff may have forgotten to do the testing or decided not to conduct lipid screening based on other variables that were not measured in this study, such as patient refusal or presence of comorbidities. Secondly, not all patients received a full lipid screen. Lipids that were ordered on the ward were ordered at the doctor's discretion rather than on a standard form, whereas lipid orders from the ED were mostly requested on a standard pathology form that included TC, triglycerides, LDL-C, HDL-C, and Apo B. Thirdly, although the study was conducted prospectively, some data were gathered via medical record review, which has a number of inherent limitations including missing information and poor documentation, all which compromise the quality of the data set. Finally, the high dyslipidaemia prevalence in the study sample still may be reflective of a high prevalence in the general community treated at the study hospital in contrast with the rest of Australia. We did not test a control group of patients without chest pain and thus are unable to determine if this might be augmenting the results. It does not, however, reduce the benefit to be had from performing lipid testing in such patients when they present with chest pain.

CONCLUSION

A moderate, but useful, increase in the detection rates of dyslipidaemia is possible if lipid testing is offered to all patients presenting with chest pain, rather than only those who are admitted to wards for further investigations and management of suspected acute coronary syndromes. Testing of this group should be considered as a health promotion initiative in the ED, with appropriate follow up in the community.

ACKNOWLEDGEMENT

We thank P Maddy, Senior Scientist in Biochemistry at Western Hospital for his extensive assistance with the pathology database aspects of this study.

Authors' affiliations

C V Elder, D Kerr, A-M Kelly, Joseph Epstein Centre for Emergency Medicine Research and the University of Melbourne, Australia
R X Davey, Pathology Service, Western Hospital, Australia

Competing interests: none declared

REFERENCES

1 Australian Institute of Health and Welfare 2002. *Australia's Health 2002*. Canberra: Australian Institute of Health and Welfare, 2002:9–118.
2 **National Heart Foundation of Australia, Cardiac Society of Australia and New Zealand**. Lipid management guidelines—2001. *Med J Aust* 2001;**175**(suppl):S57–S85.
3 **McElduff P**, Dobson AJ, Jamrozik K, *et al*. Opportunities for control of coronary heart disease in Australia. *Aust N Z J Public Health* 2001 Feb; **25**:24–30.
4 **Burns RB**, Stoy DB, Feied CF, *et al*. Cholesterol screening the emergency department. *J Gen Intern Med* 1991;**6**:210–15.
5 **Diercks DB**, Kirk JD, Turnipseed SD, *et al*. Cholesterol screening in an ED-Based chest pain unit. *Am J Emerg Med* 2002;**20**:510–12.
6 **Chandra A**, Compton S, Sochor M, *et al*. Untreated hypercholesterolemia in an emergency department chest pain observation unit population. *Acad Emerg Med* 2002;**9**:699–702.
7 **Friedewald WT**, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 1972;**18**:499–502.
8 **Bhatnagar D**, Durrington PN. Measurement and clinical significance of apolipoproteins A-1 and B. In: Rifai N, Warnick GR, Dominczak MH, eds. *Handbook of lipoprotein testing*, 2nd edn. Washington DC: American Association for Clinical Chemistry, 2000:287–310.
9 **National Cholesterol Education Program: Second report of the expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel II)**. *Circulation* 1994;**89**:1333–445.
10 **Reinhart RA**, Gani K, Arndt MR, *et al*. Apolipoproteins A-I and B as predictors of angiographically defined coronary artery disease. *Arch Intern Med* 1990;**150**:1629–33.
11 **The Australian Diabetes, Obesity and Lifestyle Study (AusDiab)**. In: Dunstan D, ed. *Diabesity and associated disorders in Australia—2000: the accelerating epidemic*. Melbourne: International Diabetes Institute, 2001.
12 **Royal Australian College of General Practitioners**. National Preventive and Community Medicine Committee. Guidelines for preventive activities in general practice. *Aust Fam Physician* 2001;**30**(special issue):S1–S61.