Secondary prevention following myocardial infarction: evidence from an audit in South Wales that the National Service Framework for coronary heart disease does not address all the issues

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ORIGINAL ARTICLE

Coronary heart disease (CHD) is an important cause of death and serious morbidity in the UK. In March 2000 the Government published their National Service Framework (NSF) for CHD which outlined future CHD healthcare provision. The secondary prevention drug targets for CHD are shown in Box 1. An early priority is to increase the effectiveness of these drugs in CHD prevention established CHD. As large studies have established the converting enzyme (ACE) inhibitors to 80–90% of patients with heart failure. However, follow up indicated problems in ongoing care with cholesterol targets not always being achieved, β blockers often being stopped, and ACE inhibitors frequently remaining at low doses. Gaining maximum benefit from treatment depends on these secondary targets also being achieved. In these aspects of secondary prevention the NSF represents only an initial step towards effective prevention of coronary heart disease; perhaps the most difficult and expensive steps are yet to be fully realised.

METHODS

Llandough Hospital, Cardiff is a 500 bed teaching hospital with unselected medical intake. Coronary patients are managed in a five bed coronary care unit by general physicians with backup from two visiting cardiologists (from the nearby University Hospital of Wales). Each acute admitting team consists of two consultants, two specialist registrars (SpRs), two senior house officers (SHOs), and two house officers (HOs).

Objectives: To assess local uptake of treatments for secondary prevention of myocardial infarction and compare with targets in the National Service Framework (NSF) for coronary heart disease.

Design: Retrospective audit of case notes with follow up questionnaire at 1 year.

Setting: Teaching hospital and community.

Participants: 100 patients alive in December 1998 who had been admitted with an acute myocardial infarction between October 1997 and October 1998.

Main outcome measures: Local use of aspirin, β blockers, ACE inhibitors, and statins.

Results: Unless contraindicated, discharge aspirin use was 100%, β blocker use 84%, statin prescription and/or provision of dietetic advice 66%, and ACE inhibitors where any heart failure was found was 97%. 1–2 years later total cholesterol remained greater than 5.0 mmol/l in 25% of patients, 24% had stopped β blockers, and ACE inhibitors remained at a low dose in half of those surveyed.

Conclusions: The NSF for coronary heart disease states that by April 2002 80–90% of patients should be prescribed appropriate secondary prevention. This had nearly been achieved at hospital discharge in 1999. However, follow up indicated problems in ongoing care with cholesterol targets not always being achieved, β blockers often being stopped, and ACE inhibitors frequently remaining at low doses. Gaining maximum benefit from treatment depends on these secondary targets also being achieved. In these aspects of secondary prevention the NSF represents only an initial step towards effective prevention of coronary heart disease; perhaps the most difficult and expensive steps are yet to be fully realised.

Box 1 NSF for CHD secondary prevention drug targets

- Give low dose aspirin.
- Give β blockers for at least 1 years following myocardial infarction.
- Provide advice and treatment to maintain blood pressure below 140/85 mm Hg.
- Give statins to lower serum cholesterol concentrations either to <5 mmol/l (LDL-C to <3 mmol/l) or by 30% (whichever is greater).
- Give ACE inhibitors to those with symptomatic heart failure, echocardiographic evidence of left ventricular dysfunction, or extensive Q wave infarcts.
- Control glucose levels meticulously as well as blood pressure in patients who also have diabetes.

Initial assessment

Audit standards were set from a review paper published by Mehta and Eagle (see box 2) and were agreed locally before the start of the study (November/December 1998). No similar audit had been conducted within the trust before 1998 and no CHD management guidelines were in use. Data collection was retrospective from case notes by four doctors (two HOs, one SHO, and one SpR) using an in-house proforma (December 1998–January 1999). The SpR was able to resolve any difficulties in data collection. Included patients had to have been alive at the start of the audit and discharged with a new diagnosis of acute MI between 1 October 1997 and 31 October 1998. ICD-10 discharge codes 121.0–121.4 and 121.9 were used for...
selection. All case notes were requested at the same time but, to minimise costs, we used the first 100 to arrive in the department.

Follow up
In October 1999 all surviving locally resident patients (identified using the hospital computer system) were sent a postal questionnaire requesting a medication list with doses. All were asked whether they were taking daily aspirin and, if not, whether they knew why not. Non-responders were sent a reminder questionnaire.

Recent total cholesterol results were obtained from the hospital computer system.

RESULTS
A total of 183 patients were discharged from Llandough Hospital following an acute MI between October 1997 and October 1998; 100 (55%) were audited (63% male; age range 41–89 years; 47% over 70 years of age). By October 1999 10 patients had died and three were living outside the area; 75 (41–89 years) had a total cholesterol level which exceeded 5.0 mmol/l; 24 of these (31%) were referred to a dietician and 42 (55%) were discharged on a statin (93%) patients. Seventy seven (83%) had a total cholesterol level in excess of 5.0 mmol/l. Only three patients under 70 years with a total cholesterol level greater than 5.0 mmol/l were not on a statin in October 1999. However, of those who reported statin use in 1999, eight (18%) still had a total cholesterol level of more than 5.0 mmol/l.

DISCUSSION
Secondary prevention of acute MI has been the subject of several reviews and large surveys. Our use of aspirin, β blockers, statins, and ACE inhibitors was better than previously reported, but the small sample size and non-random selection precludes direct comparisons. The NSF for CHD sets the target for use of secondary prevention treatments following acute MI at 80–90% to be achieved by April 2002. Our findings indicate that some of these targets were achieved in our area by 1999 but, more importantly, we have highlighted several problems with ongoing patient care which were poorly considered in the NSF.

One quarter of patients had discontinued β blockers by the time of the follow up. The benefits of β blockade following an acute MI have recently been re-emphasised and there is good evidence that patients on β blockers, irrespective of actual or perceived contraindications, do better than those not receiving them. We do not have detailed information about each case, but the trend underlines a problem with β blocker treatment. Patients are reluctant to take them and many doctors stop them at the first hint of a contraindication.

Although we were successful in initiating ACE inhibitors in patients with signs of heart failure, many remained on doses lower than those used in clinical trials. Avoidance of early complications of ACE inhibitors necessitates close supervision during initiation and upward titration. Although safe in the community, every effort should be made to increase treatment as an inpatient. With additional evidence favouring early ACE inhibition (0–36 hours) following an acute MI, this is not unrealistic although the decreasing length of hospital stay does create problems with dose escalation.

Of all the treatments proved to be successful in CHD prevention, the underuse of statins has been the most widely reported. Although our follow up indicates that many patients were prescribed these drugs by October 1999, at least one fifth needed further dose titration. The elderly accounted for most of those not receiving treatment by 1999. Elderly patients have a high absolute risk of CHD and are likely to benefit significantly from appropriate cholesterol lowering treatment.

If we are to realise the full potential of these drugs for CHD prevention, we need to ensure they are used to maximal effect. We have shown that achieving high prescribing levels is only a first step, and possibly the easiest to implement with suitable training. Important further targets are the appropriate continuation of β blockers, achievement of ACE inhibitor dosage goals, and satisfactory cholesterol levels. How best to deliver these aspects of continuing CHD care is not discussed.
in the NSF document. Additional resources are needed to ensure adequate patient follow-up both in hospital and general practice. This could take the form of additional hospital-based CHD clinics, perhaps nurse led, or a consultant/general practice run liaison service linking primary and secondary care. This is especially true in our area where the transition from hospital back to the community needs better integration around the time of hospital discharge. Improved communications between doctors and better patient education at this crucial time are also likely to be important issues.

Highly effective CHD prevention may yet be a bigger undertaking than is suggested in the NSF. Unless sufficient resources become available for treatment optimisation and better lines of communication are established between secondary and primary care, the achievement of these NSF targets may not necessarily equate with more effective patient care.

Key messages

- The National Service Framework for coronary heart disease is an important document outlining future plans for heart disease prevention and treatment.
- 80–90% of those with established coronary heart disease should be prescribed aspirin, β blockers, statins, and ACE inhibitors by April 2002.
- Achieving NSF secondary prevention prescribing targets offers only a beginning. For truly effective prevention of coronary heart disease we must also ensure adequate therapeutic effect and prescription continuation over many years.

REFERENCES


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doi: 10.1136/qhc.11.3.230

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