The use of HMG Co-A reductase inhibitors following acute myocardial infarction in hospital practice

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Summary

Treatment with a HMG-CoA reductase inhibitor (statin) following a myocardial infarction has been shown to reduce the incidence of subsequent coronary revascularisation, myocardial infarction and cardiovascular death. The majority (89%) of patients admitted to the coronary care unit of our hospital received a fasting cholesterol check as part of a routine coronary care unit protocol. However, our survey shows that only 26% of patients surviving an acute myocardial infarction were on treatment with a statin at follow-up. Furthermore, those receiving statins were given smaller doses than those used in clinical trials. One way to ensure patients receive adequate treatment with statins, may be to include it as part of a coronary care unit protocol.

Keywords: statins; myocardial infarction

Several well-controlled trials in patients with coronary heart disease have shown that the long-term use of hydroxymethylglutaryl coenzyme A (HMG Co-A) reductase inhibitors ("statins") causes a reduction in the incidence of acute myocardial infarction (AMI) and death from cardiovascular causes, and substantially reduces the need for coronary revascularisation.1-3 We decided to perform a small pilot study in our hospital to see how many patients discharged following AMI were receiving a statin.

Methods

We performed a retrospective analysis of all case notes of patients discharged from the coronary care unit (CCU) of our hospital with a definite diagnosis of AMI between January and December 1996. Our aim was to assess whether treatment with lipid-lowering therapy was prescribed at the time of discharge from hospital or at follow-up. The results are expressed as mean ± standard deviations.

Results

Of the 116 case notes requested, 101 were available for analysis (mean age 64 ± 12 years; 69 males), of whom 71 patients were Caucasian, 25 Asian and five Black. The site of AMI was as follows: 46 anterior, 46 inferior, four posterior and five others. Forty-three of these patients had been receiving treatment for hypertension, 23 for diabetes mellitus, 32 had a documented history of previous coronary heart disease (angina or previous AMI) and 80 were current or ex-smokers. One patient was already on treatment with lipid-lowering therapy (pravastatin 10 mg once daily).

A fasting or random cholesterol level was performed on admission to casualty or on the morning following admission to the CCU in all but 11 (11%) patients (mean cholesterol level 5.8 ± 1.2 mmol/l). At the time of discharge from hospital, 13 (13%) patients were receiving treatment with a statin (mean pre-treatment cholesterol level 6.8 ± 0.7 mmol/l; mean age 59 ± 12 years). Twelve were receiving simvastatin (mean daily dose 13 ± 5 mg), and one pravastatin 10 mg daily. The fasting cholesterol level in patients who did not receive treatment with a statin was 5.6 ± 1.1 mmol/l (42 of these 88 patients had a fasting cholesterol greater than 5.5 mmol/l; mean age 64 ± 13 years).

After a mean out-patient follow-up of 5 months, an additional 10 patients were found to be receiving a statin. Thus, in total, 22 patients were receiving treatment with a statin; 20 with simvastatin (mean dose 13.2 ± 4.8 mg daily; 13 subjects on 10 mg daily, seven on 20 mg daily), one with pravastatin 10 mg daily, and one with fluvastatin 40 mg daily. Thirteen patients, according to the notes, either did not attend or were lost to hospital follow-up. Therefore, a total of 26% (23 subjects) of the 88 who attended follow-up were being treated with a statin.

Conclusions and discussion

Our results show that, in 1996, of 101 patients who survived a myocardial infarction at St George’s Hospital, only 13% were being treated with a statin at discharge and 26% at follow-up. This is surprising in view of the overwhelming evidence that long-term use of statins reduces fatal and non-fatal events in subjects with ischaemic heart disease; and also reduces the need for coronary revascularisation. For example, the 4S Study suggested that treatment of 100 patients with coronary artery disease with simvastatin, 20 to 40 mg daily, would result in the prevention of death in four out of the nine expected deaths from coronary heart disease over 6 years.4

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Our study also shows that, in the few patients who were given a statin, many were on a lower dose than those shown to be effective in clinical trials. It is clearly a matter of controversy as to what level of cholesterol should be achieved but until trials have been done with lower doses, it would seem prudent to use doses similar to those which have proved effective in clinical trials. The more recent CARE Study demonstrated that benefit can be gained from treating patients with coronary heart disease at lower cholesterol levels (mean of 5.3 mmol/l) with pravastatin, which indicates that lipid-lowering therapy should be based on overall cardiovascular risk rather than the level of cholesterol. In other words, patients who have had an AMI are in a high risk group and should all receive a statin unless there is a contraindication.

It is possible that statins have other benefits independent of their cholesterol-lowering effect and some studies have suggested an improvement in thrombogenic risk and endothelial function in patients with hypercholesterolaemia treated with pravastatin. One encouraging finding from our survey was that nearly all patients admitted to the CCU had their cholesterol measured, as this was part of the CCU protocol. Therefore, the best way to ensure that all patients surviving an AMI receive a statin would be to make it part of the CCU protocol, so that all patients were commenced on a statin during their hospital stay.

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