Effects of self monitoring of triglyceride concentrations in non-insulin dependent diabetes

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BMJ 1993;306:493

Cardiovascular disease is three times more common in diabetic patients than the non-diabetic population and is the main cause of death in people with non-insulin dependent diabetes mellitus.¹ The importance of the plasma triglyceride concentration as a risk factor for coronary heart disease in the general population is still uncertain. In diabetic patients, however, triglyceride concentrations are an important predictor of coronary heart disease,² and patients with non-insulin dependent diabetes are twice as likely as non-diabetic people to be dyslipidaemic,³ hypertriglyceridaemia being the commonest lipid abnormality.⁴ As diabetic patients have little feedback on their daily lipid concentrations we aimed at determining whether self monitoring of plasma triglyceride concentrations improved lipid concentrations (and perhaps indirectly glucose concentrations) in patients with non-insulin dependent diabetes mellitus.

Methods and results

Twelve non-insulin dependent diabetic patients with fasting plasma triglyceride concentrations >1.7 mmol/l were recruited from diabetic clinics in Newcastle upon Tyne. The local ethical committee's approval and patients' informed consent were obtained for the study. No patients had fasting triglyceride concentrations >5.0 mmol/l or cholesterol concentrations >7.8 mmol/l, none were receiving lipid lowering treatment or insulin, and all had a haemoglobin A_{1c} concentration <9.5 (reference range 2.8-4.4)% and a body mass index <35 kg/m². At the time of recruitment the patients were reminded of the British Diabetic Association's dietary recommendations (55% carbohydrate, 30% fat, and 15% protein).⁵ The patients were divided into two groups matched for age, sex, body mass index, and fasting plasma triglyceride concentrations. Patients in one group were given Reflotron machines (Boehringer Mannheim, Germany) and were asked to measure their triglyceride concentrations at home, before meals and before going to bed, twice a week and to record the results in diaries. The other group acted as controls. All patients attended the clinic monthly for six months. They fasted before each visit, when they were weighed and had blood samples taken for measurement of triglyceride, cholesterol, high density lipoprotein cholesterol, apolipoprotein B, and haemoglobin A_{1c} concentrations.

The mean plasma triglyceride concentration of the patients who monitored themselves fell significantly during the study (2.7 (SD 0.7) mmol/l at the start *v* 1.6 (SD 0.4) mmol/l at the end; difference = 1.1 (95% confidence interval -0.1 to 2.3), as did their plasma cholesterol concentration (6.6 (0.9) *v* 6.1 (0.7) mmol/l; difference = 0.5 (-0.3 to 1.3) and apolipoprotein B concentration (1.18 (0.19) *v* 0.96 (0.17) g/l; difference = 0.2 (-0.12 to 0.52). There was no significant change in haemoglobin A_{1c} concentration (6.3 (1.4) *v* 5.8 (2.4)%; $p=0.4$) or body weight (77 (17) *v* 75 (20) kg; $p=0.3$). Among the controls no significant changes occurred in any of the variables measured.

The accuracy of patients' self monitoring was shown by the good reproducibility of measurements of control solutions (Precinorm, Boehringer Mannheim) and by the good correlation between the monthly triglyceride concentrations measured in the laboratory and the mean monthly preprandial values measured at home ($r=0.88$, $p<0.001$). There was a good correlation between individual triglyceride concentrations measured preprandially and at bedtime ($r=0.84$, $p<0.001$). Three months after the end of the study triglyceride concentrations had risen in the patients who had monitored themselves from 1.6 to 2.2 mmol/l (NS), as had the patients' cholesterol concentrations (from 6.1 to 6.4 mmol/l (NS)).

Comment

In this pilot study we showed that self monitoring of hypertriglyceridaemia in patients with non-insulin dependent diabetes mellitus improved lipid profiles over six months. Among patients using blood testing for glycaemic control this may be a useful adjunct to self management, giving feedback, encouraging adherence to a low fat diet, and possibly allowing lipid lowering treatment to be avoided. A larger study is required to establish whether improvement can be sustained.

We thank Boehringer Mannheim for financial support.

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(Accepted 16 December 1992)

Estimating urinary albumin excretion rate of diabetic patients in clinical practice

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BMJ 1993;306:493-4

An increase in urinary albumin excretion is recognised as predicting impending renal failure in diabetic patients and cardiovascular disease in the general population.¹ Measurement of the albumin excretion rate requires an accurately timed collection of urine, which is difficult in routine clinical practice. The

albumin excretion rate correlates well with both the albumin concentration and the ratio of albumin to creatinine concentrations in first morning urine specimens.² It has therefore been accepted that the urinary albumin concentration or albumin:creatinine ratio can be used to screen patients.¹

Before the clinical importance of such variables can be assessed the variability in a patient's urinary albumin concentration and albumin:creatinine ratio must be known. Data on individual variability have been published for small numbers of patients and controls, and coefficients of variation have been 31-43% under study conditions.^{3,4} This study was designed to establish intraindividual variability in urinary albumin concentrations and albumin:creatinine ratios under routine clinical conditions.