Short Report - Organisation and Delivery of Care

Glycaemic control and lipid concentrations in a cohort of people with diabetes over 7 years of follow up: a regional audit of diabetes care in the UK

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Bulleted novelty statement:

- This is the first report of a longitudinal audit of the attainment of glycaemic and lipid targets in a large cohort of individuals with diabetes followed for 7 years, after the introduction of the Quality and Outcomes Framework targets in the UK.
- The audit shows surprising maintenance of glycaemic control throughout the 7 year period of the audit with a mean HbA_{1C} of 62.1 mmol/mol ± 16.1 in 2006, compared to 61.7 mmol/mol ± 17.3 in 2013.
- There was improvement in the proportion of people achieving target total cholesterol of < 5 mmol/L from 79% in 2006 to 83% in 2013

Abstract = 250 words

Aim: To determine changes in glycaemic control and lipids over time since the introduction of the Quality and Outcomes Framework (QOF).

Methods: In adults with diabetes (Hampshire, UK), HbA_{1C} and lipid measurements were retrieved from a regional NHS biochemical database in 2006 and 2013 and analysed by ANOVA and logistic regression modelling.

Results: In 2006, 8568 people with diabetes were identified. In 2013, 5815 had follow up data, 1207 people were lost to follow-up and 1546 had died. At baseline, HbA_{1C} was 62.1 ±16.1, 64.7 ±16.7 and 64.5 ± 17.6 mmol/mol; for those with follow up data, lost to follow up and those who died, respectively. Mean age was 60.2±14.5, 57.6 ± 18.0 and 73.9±10.5 years respectively for the three groups. Total cholesterol, HDL-C and triglyceride concentrations were similar between groups.

Mean HbA_{1C} for those with complete follow-up was 62.1±16.1 in 2006 and 61.7 ±17.3 mmol/mol in 2013. QOF targets for cholesterol (<5 mmol/L) were achieved by 79% in 2006 and 83% in 2013 (P<0.001). Baseline age and HbA_{1C} were associated with death at follow up; odds ratio (OR) per year increase in baseline age 1.10 (95% CI 1.09-1.10, P<0.001), and per unit increase in HbA_{1C} OR 1.02 (95% CI 1.02-1.03, P<0.001).

Conclusions: Glycaemic control showed remarkable stability over 7 years follow-up, despite increasing patient age and duration of diabetes. More patients achieved lipid targets in 2013 versus 2006. Although baseline HbA_{1C} was a predictor of death at follow-up, baseline HbA_{1C} differed little between survivors, non-survivors and those lost to follow-up.

Introduction

The global prevalence of type 2 diabetes is increasing and in the UK it is predicted that 5 million people will have diabetes by 2025 [1]. It is well recognised that type 2 diabetes is a progressive disorder with a tendency to worsening glycaemic control over time [2]. Data from the UK Prospective Diabetes Study showed that there is an inexorable decline in glycaemic control in people with newly diagnosed type 2 diabetes over time despite treatment [3], and the data showed that HbA_{1C} tends to increase by around 11mmol/mol (1%) every 2 years [4]. In contrast, approximately 10% of adults with diabetes in the UK have type 1 diabetes, and it has been shown that glycaemic control remains relatively stable in this condition [5].

LDL-cholesterol (LDL-C) is an independent risk factor for CVD [6] and since treatment with statins produces a 25-40% risk reduction in cardiovascular events [7-9], the National Institute for Health and Care Excellence (NICE) has recommended treatment of total cholesterol in people with diabetes to <4mmol/L (or LDL-C <2mmol/L) and recommends a cut-off of 5mmol/L for audit purposes [10].

The Quality and Outcomes Framework (QOF) was introduced in April 2004 [11]. This scheme offers performance-based pay incentives for achieving various outcome measures related to glycaemic control and cholesterol concentrations in people with diabetes. Diabetes is the largest single clinical area within the QOF, although the impact that this initiative has had on the quality of diabetes care in the UK is unclear [12][13].

With the introduction of QOF, improved awareness and better treatments for hyperglycaemia, we have tested whether there is a deterioration in HbA_{1C} in patients with diabetes by following a cohort of patients with diabetes over 7 years between 2006 and 2013. We have also investigated for the possibility of survival/selection bias affecting our results by analysing baseline data for all patients, including those who died and those who were lost to follow up.

Subjects and methods

A retrospective observational audit of 8568 adults with diabetes followed-up over 7 years between 2006 and 2013 was undertaken. In 2006, 8568 people with diabetes were identified by virtue of their having had an HbA1c requested. It was usual practice in our region at that time to only request an HbA1c measurement in patients with established diabetes. 5815 had follow-up data available in 2013. We audited HbA_{1C}, total cholesterol, HDL-cholesterol and triglyceride results from 2006 and 2013.

Statistical analysis was undertaken using SPSS 22 (SPSS Inc., Chicago, IL, USA). Between group differences in baseline HbA_{1C} and lipid measurements, for patients with follow up data, those who were lost to follow-up, and those who were known to have died was analysed by analysis of variance (ANOVA). We compared means for HbA_{1C} and total cholesterol for 2006 and 2013 using paired T-tests. Proportions of patients achieving QOF targets for HbA_{1C} and total cholesterol were compared using Chi-squared tests. Binary logistic regression was used to determine whether baseline measurements were associated with proven vital status at follow-up and loss-to-follow-up in 2013. Multivariable linear regression modelling was used to determine whether baseline measurements in 2006 were associated with HbA_{1C} at follow up in 2013.

Caldicott principles were adhered to in the conduct of the audit. Approval of the audit and its subsequent publication was granted by the Caldicott Guardian at University Hospital Southampton.

Results

Figure 1 illustrates the number of patients initially identified in 2006 for the study, how many had follow-up data and how many had died or were otherwise lost to follow-up by 2013.

Data in Table 1 show mean HbA_{1C}, total cholesterol, HDL-cholesterol and triglyceride concentrations in 2006 and 2013 in the same people in whom data were available at both time points, as well as comparisons of proportions of patients achieving QOF targets. Mean HbA_{1C} remained virtually the same in 2006 compared to 2013 (HbA_{1C} 62.1±16mmol/mol (7.8%±2) vs 61.7±17 (7.8%±2.1)). A small reduction in mean total cholesterol was observed

in 2013 from 2006 (4.4±1mmol/L to 4.1±1). There was a small increase in the proportion of patients achieving the QOF target of HbA_{1C} <58mmol/mol (7.5%) in 2006 compared with 2013 (47 vs 49%). There was a significant marked increase in the proportion of people reaching QOF targets for total cholesterol and for HbA_{1C} combined between 2006 and 2013 (37 vs 42%).

Table 1 also shows the differences in baseline 2006 characteristics between those with full follow-up data, those who went on to die prior to 2013, and those who were lost to follow-up. At baseline, patients with full follow-up data had a mean±SD age of 60.7±13.7 years compared with 73.9±10.5 years for those who died and 57.6±18 years for those lost to follow-up. Mean HbA_{1C} in 2006 was 62±16mmol/mol (7.8%±2) for those with full follow-up data, 65±18 (8.1%±2.2) for those who died during follow-up and 65±17 (8.1%±2.1) for those who were lost to follow-up.

Baseline age, HbA_{1C}, total cholesterol, HDL-C and triglycerides were entered into a binary logistic regression model with vital status in 2013 as the outcome. Baseline age and HbA_{1C} were associated with death at follow up; adjusted odds ratio (OR) per year increase in age at baseline was 1.10 (95% CI 1.09-1.10, P<0.001), and per mmol/mol increase in HbA_{1C} adjusted OR was 1.02 (95% CI 1.02-1.03, P<0.001). Increasing age was associated with a higher risk of being lost to follow-up (adjusted OR per year increase in age = 0.96, 95% CI 0.95-0.96, P<0.001) as was being female (adjusted OR 0.76, 95% CI 0.68-0.86, P<0.001) and having a higher baseline HbA_{1C} (OR per unit increase in HbA_{1C} 0.98, 95% CI 0.98-0.99, P<0.001).

Discussion

These data from a longitudinal study demonstrate surprising marked stability of glycaemic control over 7 years. We observed a marked improvement in the proportion of people achieving HbA_{1C} <58mmol/mol (7.5%) and total cholesterol of ≤5mmol/L with the proportion of people achieving both targets combined increasing from 37 to 42% between 2006 and 2013 (P<0.001).

In the National Diabetes Audit (NDA) report for 2012/2013 [14], the proportions of people with type 2 diabetes achieving the HbA_{1C} target of ≤58mmol/mol (7.5%) were 66.5%, 65.8% and 64.8% for 2010/2011, 2011/2012, and 2012/2013 respectively. These percentages are higher than those reported in our audit, but we are unable to discriminate between people with type 1 and type 2 diabetes in our laboratory database.

There are strengths and limitations with our study design. A major strength of our study is that we have studied glycaemic control in the same individuals over seven years of follow up. . Thus, our study is not affected by ascertainment bias introduced by changing behaviour amongst health care professionals who now identify incident cases of diabetes earlier in the disease process (compared to the situation in the UK in the past). Patients who are identified early are much more likely to have better glycaemic control than those identified later in the disease process. Consequently, the results of serial population-based cross-sectional studies of secular trends in glycaemic control over time, could produce misleading results about secular trends in glycaemic control in patients with diabetes in the England and Wales. Although we have noted that baseline glycaemic control and lipid indices in 2006 were remarkably similar in people who had full follow-up data in 2013, it is uncertain how this relates to possible intensification of drug therapies and how this might relate to increasing prescribing spend [15]. A potential limitation of our study design is also the issue of survival/selection bias, due to people dying or moving out-of-area. However, the only marked difference in baseline characteristics between subjects in these different groups was age; e.g. the group that died during follow-up had a higher mean age in 2006.

We investigated the possibility of bias by using a binary logistic regression model that included vital status at follow-up as the outcome and each of the baseline factors as exposures. These data showed that baseline age was by far the strongest factor associated with death at follow-up (with only a small contribution from baseline HbA_{1C}). However, with the limited information available in our routine NHS biochemical database, it is possible that there are other unrecorded factors that may confound this analysis. It is possible that there is an association between failure to be followed up and low adherence to other health related behaviours that are not measured in our cohort and this could confound our analyses.

In conclusion, our retrospective longitudinal study of 5815 patients over a period of 7 years shows remarkable stability of glycaemic control, despite patients being seven years older and despite seven years longer diabetes duration. An increased proportion of patients are now being treated to QOF targets for HbA_{1C} and total cholesterol combined in 2013, compared with 2006. Whether implementation of QOF has played a role in the improvement in these risk factors is uncertain and whether this stability of glycaemic control and improvement in cholesterol produces a decrease in microvascular and macrovascular outcomes in people with diabetes now needs to be determined.

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Figure 1 – Flowchart to demonstrate numbers of patients included in the study and analysis of data.

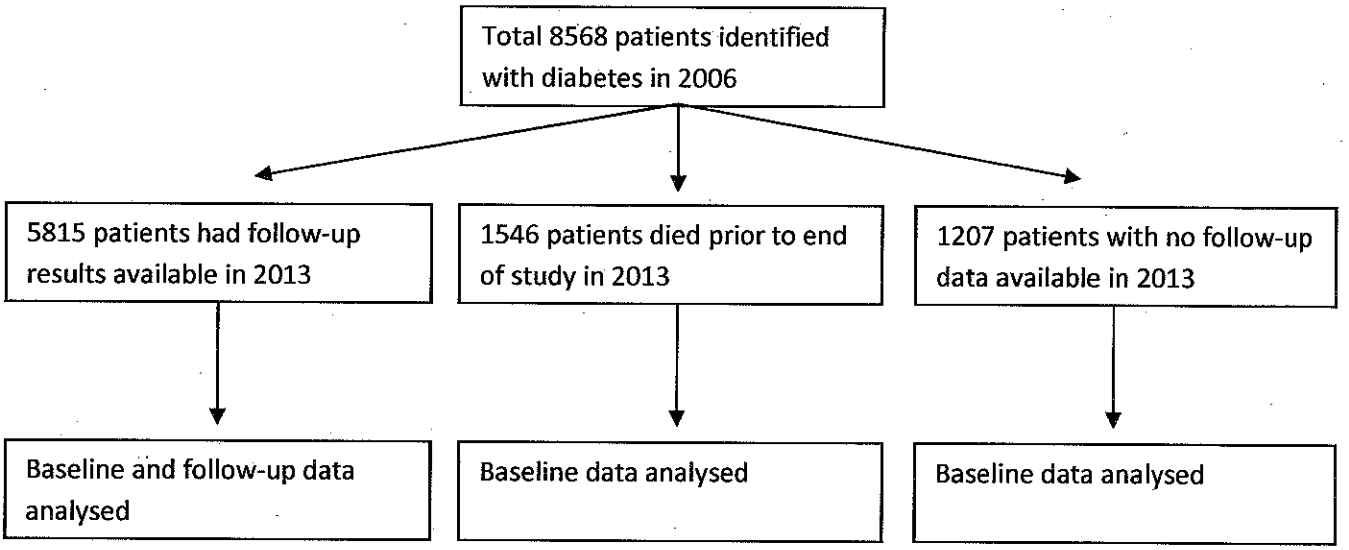

Table 1. Glycated haemoglobin (HbA_{1c}) and lipid concentrations, and proportions of patients meeting glycaemic and lipid targets for subjects with data available in both 2006 and 2013, and baseline characteristics of groups lost to follow-up.

	Complete follow-up n = 5815 (data available in 2006 and 2013)		People dead in 2013 n = 1546 (no data available in 2013)	Lost to follow-up post-2006 n = 1207 (no data available in 2013)	xP
Age (y)	60.6 ± 13.7		73.9 ± 10.5	57.6 ± 18	<0.001
% men	54		55	56	0.5
HbA_{1c} (mmol/mol (%))	**2006**	62.1 ± 16 (7.8%±2)	65 ± 18 (8.1%±2.2)	65 ± 17 (8.1%±2.1)	<0.001
	2013	61.7 ± 17 (7.8%±2.1)			
	P	0.002			
HbA_{1c} <58 mmol/mol (7.5%)	**2006**	47 %	44 %	42 %	0.006
	2013	49 %			
	P	0.045			
HbA_{1c} >86 mmol/mol (10%)	**2006**	8 %	12 %	12 %	<0.001
	2013	9 %			
	P	0.07			
Total cholesterol mmol/l	**2006**	4.4 ± 1	4.3 ± 1	4.6 ± 1.1	<0.001
	2013	4.1 ± 1			
	P	<0.001			
Total cholesterol ≤ 5.0 mmol/l	**2006**	79 %	78 %	70 %	<0.001
	2013	83 %			
	P	<0.001			
HDLc (mmol/l)	**2006**	1.20 (1.19 -1.21)	1.23 (1.20 - 1.25)	1.23 (1.21 - 1.25)	0.001
	2013	1.26 (1.25-1.27)			
	P	<0.001			
Triglyceride (mmol/l)	**2006**	1.86 (1.82 – 1.90)	1.80 (1.73 – 1.87)	1.99 (1.86 – 2.12)	0.012
	2013	1.77 (1.72-1.81)			
	P	0.029			
HbA_{1c} <7.5 and total cholesterol ≤ 5.0 mmol/l	**2006**	37 %	35 %	31 %	<0.001
	2013	42 %			
	P	<0.001			

Data are mean ± standard deviation (SD) for HbA_{1c} and total cholesterol concentration and geometric means and 95% confident intervals for high density lipoprotein cholesterol (HDL_c) and triglyceride concentrations.

P values represent differences between 2006 and 2013

xP values represent between group differences for baseline 2006 data for the group with full follow-up data, the group that died, and the group lost to follow-up

Supplementary Table 1 - Percentage of patients (by sex) who achieved QOF targets in 2006 and 2013.

2006, n = 5815			2013, n = 5815		
	sex	**%**		**sex**	**%**
HbA_{1C} <58 mmol/mol (7.5%)	M	47	**HbA_{1C} <58 mmol/mol (7.5%)**	M	50
	F	47		F	47
P		0.93			0.08
	sex	**%**		**sex**	%
HbA_{1C} >86 mmol/mol (10%)	M	7	**HbA_{1C} >86 mmol/mol (10%)**	M	8
	F	9		F	10
P		0.019			0.06
	sex	%		**sex**	%
Total cholesterol ≤5.0 mmol/L	M	82	**Total cholesterol ≤5.0 mmol/L**	M	76
	F	74		F	88
P		<0.001			<0.001
	sex	%		**sex**	%
HbA_{1C} <58 mmol/mol (7.5%) and total cholesterol ≤ 5.0 mmol/L	M	39	**HbA_{1C} <58 mmol/mol (7.5%) and total cholesterol ≤ 5.0 mmol/L**	M	46
	F	35		F	38
P		0.001			<0.001

P value represents differences between males and females within the same cohort of patients from 2006 to 2013.

Supplementary Table 2 - Percentage of patients (by age group in years) achieving QOF targets in 2006 and 2013.

Age band (years)	Year	HbA_{1c} <58 mmol/mol (7.5%)	HbA_{1c} >86 mmol/mol (10.0%)	Total cholesterol ≤5.0 mmol/L	HbA_{1c} <58 mmol/mol (7.5%) and total cholesterol ≤5.0
18 -39	2006	32	16	65	18
n = 448	2013	24	20	66	16
P		0.007	0.12	0.66	0.39
40-54	2006	37	13	73	27
n = 1252	2013	36	13	79	30
P		0.43	0.64	0.001	0.13
55-69	2006	46	7	81	38
n = 2439	2013	52	8	85	46
P		<0.001	0.48	<0.001	<0.001
70-84	2006	58	4	83	49
n = 1626	2013	60	5	86	54
P		0.24	0.31	0.005	0.007
>84	2006	62	0	74	44
n = 50	2013	60	6	73	46
P		0.84	0.08	0.91	0.86

P value represents differences in proportions of patients achieving targets for 2006 and 2013 by age group.