Trends in the smoking habits of young adults with diabetes

I MacFarlane, G Gill, T Grove, M Wallymahmed

Abstract

Objectives—To examine changes in the prevalence of smoking in young adult diabetic patients between 1990 and 1999.

Setting—Walton Diabetes Centre, University Hospital Aintree, Liverpool, UK.

Design—Direct questioning as well as the urinary cotinine:creatinine ratio were used to assess the smoking habits of 99 young type 1 diabetic patients in 1991 (mean age 21.5 years, duration of diabetes 7.3 years), and in 112 similar patients in 1999 (mean age 23.4 years, duration of diabetes 9.6 years).

Results—The admitted smoking rate was 31/99 (31%) in 1990 compared with 31/112 (28%) in 1999 (not significant). However, in 1990 there were an additional 17 “covert” smokers (patients who denied smoking, but had an unequivocally raised urinary cotinine:creatinine ratio), but only three in 1999 (p<0.05). This gave a corrected validated smoking rate of 48/99 (48%) in 1990 and 34/112 (30%) in 1999, representing a significant fall (p<0.02).

Conclusion—Smoking rates in young type 1 diabetic patients appear to have fallen during the last decade, and reporting of smoking behaviour is now more honest.

Keywords: smoking; diabetes mellitus; type 1 diabetes; cotinine

Many young people with diabetes start smoking and once started have great difficulty quitting, even though the great majority wish to stop. A diabetic patient who smokes has a greatly increased risk of developing ischaemic heart disease compared with a non-smoking non-diabetic person.1 2 Smoking also increases the risk of cerebral and peripheral vascular disease and appears to aid the development and progression of microvascular disease, particularly diabetic nephropathy.1 2

In 1990 a survey of smoking habits, validated by urine cotinine concentrations, found that almost half the young type 1 adults in our hospital clinic were current smokers.3 Those who had developed diabetes in childhood or adolescence were as likely to be smokers as those who developed diabetes as young adults, indicating that antismoking counselling and education had been ineffective in the paediatric clinic. Over the past nine years, antismoking counselling/education has been given priority in our young adult clinics. Also nicotine replacement therapy is now available.4 We therefore tried to ascertain whether the prevalence of smoking had altered during this time by comparing the smoking habits of our current young adults with type 1 diabetes in 1999 with our 1990 cohort.

Patients and methods

One hundred and twelve consecutive type 1 diabetic patients attending the young adult clinic at Walton Hospital Diabetes Centre, Liverpool, were studied (60 male). Mean (SD) age was 23.4 years (4.5), range 16–30 years, and mean (SD) duration of diabetes was 9.6 (6.1) years, range 0.4–25 years. All had normal renal function. Thirty one had developed diabetes as children (0–10 years of age), 43 as adolescents (11–15 years of age), and 38 as young adults (>15 years of age). The patients who had developed diabetes in childhood or adolescence were usually transferred from the local paediatric diabetic clinic at age 16 years. In both the paediatric and young adult clinics all patients and their families received regular antismoking counselling and education by clinic doctors and nurses, warning of the adverse health effects of smoking. Posters were prominently displayed and leaflets were readily available reinforcing these issues.

Patients were asked about their current and past smoking habits at their clinic visit, and the information recorded. At the same time, the early morning urine sample they had brought to the clinic for “dipstick” testing was analysed for cotinine and creatinine, and the urinary cotinine:creatinine ratio estimated as described elsewhere.5 Urinary cotinine provides an objective measure of smoking status, as well as of nicotine “load”, over the previous approximately 48 hours. A urinary cotinine:creatinine ratio of >1 µg/mg was taken as evidence of active smoking. This level is widely accepted as effectively excluding a non-smoking status, dietary sources of cotinine, or the effects of passive smoking.6 7

The results of this survey were compared with a similar survey in 1990.3 Statistical analysis was by χ² testing with Yates’s correction and Student’s unpaired t test.

Results

The patients in the two surveys, nine years apart, were comparable for age, sex, and duration of diabetes (table 1). The claimed and actual smokers (verified by a urine cotinine:creatinine ratio >1.0) are shown for the two surveys. In 1990, 31% of patients admitted smoking compared with 28% in 1999. However when the urine cotinine:creatinine measurements were examined, 48% of the 1990 cohort were actually smoking compared with 30% of the 1999 patients, a result which was statistically significant (p<0.02). Seventeen
Table 1  Details of the smoking habits of young adults with type 1 diabetes in 1990 and in 1999, Walton Diabetes Centre, Liverpool

<table>
<thead>
<tr>
<th></th>
<th>1990 Survey (n=99)</th>
<th>1999 Survey (n=112)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male:female ratio (%)</td>
<td>53:47</td>
<td>54:46</td>
<td>NS</td>
</tr>
<tr>
<td>Age (years)</td>
<td>21.5 (3.9), range 15–31</td>
<td>23.4 (4.5) range 16–30</td>
<td>NS</td>
</tr>
<tr>
<td>Duration of diabetes</td>
<td>7.3 (5.2), range 0.9–34</td>
<td>9.6 (6.1) range 0.9–28</td>
<td>NS</td>
</tr>
<tr>
<td>Admitted smoking</td>
<td>31 (14 female) = 31.3%</td>
<td>31 (16 female) = 27.6%</td>
<td>NS</td>
</tr>
<tr>
<td>Claimed ex-smokers</td>
<td>14</td>
<td>17</td>
<td>NS</td>
</tr>
<tr>
<td>Actual smokers (cotinine verified)</td>
<td>48 (25 female) = 48.5%</td>
<td>34 (18 female) = 30.4%</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>Median age (years) started cigarettes (admitted)</td>
<td>16 (10–20)</td>
<td>16 (12–21)</td>
<td>NS</td>
</tr>
<tr>
<td>Nicotine replacement in past</td>
<td>None</td>
<td>4 (3 patch, 1 inhaler)</td>
<td></td>
</tr>
</tbody>
</table>

Note: data are expressed as means with SD in brackets. A urinary cotinine:creatinine ratio of >1 µg/mg was taken to indicate active smoking.

Table 2  Proportion of smokers by age of onset of diabetes, in 1990 and 1999 cohorts

<table>
<thead>
<tr>
<th>Age of onset of diabetes</th>
<th>1990 Survey (n=99)</th>
<th>1999 Survey (n=112)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Childhood (0–10 years)</td>
<td>9/24 (38)</td>
<td>5/31 (16)</td>
</tr>
<tr>
<td>Adolescents (11–15 years)</td>
<td>23/43 (54)</td>
<td>13/43 (30)</td>
</tr>
<tr>
<td>Young adult (16–30 years)</td>
<td>15/32 (47)</td>
<td>16/38 (42)</td>
</tr>
</tbody>
</table>

Discussion

Smoking is a major risk factor for coronary artery disease in both diabetic and non-diabetic groups. Indeed, with their greatly increased coronary risk, diabetic patients (particularly young ones) have the most to gain by either not starting smoking, or stopping the habit. All diabetic clinics should include antismoking advice as part of their care package, but the effect of such advice on the young adults in our clinic was shown to be very limited. More intensive advice, and a separate “stop smoking” clinic has also been shown to have only limited success. In view of this, it is encouraging that our results show a significant reduction in verified smoking prevalence in the past decade (48% in 1990 to 30% in 1999, p<0.02). In the 1999 cohort, fewer smokers had started in childhood and adolescence compared with the 1990 group (though the changes were not statistically significant), perhaps indicating that early advice, possibly compounded by peer and/or parental influence is now taking effect in reducing smoking uptake in young diabetic people.

Also encouraging is that the deception rate (difference between admitted smoking and positive urinary cotinine:creatinine ratio) in 1999 had significantly reduced compared to 1990 (17% v 3%, p<0.05). Previous studies have shown a high level of deception amongst young diabetic smokers. This is less of a problem in older, mainly (type 2) diabetic patients.

Smoking by close family members is more common in young diabetic patients actively smoking. Clearly, education of the whole family is important, and if parents and siblings can be encouraged to stop, this may reduce the chance of young diabetic people taking up the habit. Also, passive smoking in families is a further recognised health hazard.

Though smoking is a major additive risk factor in diabetic patients, we are aware of no comparable studies investigating changes of validated smoking habits with time in diabetic cohorts. The reduction in smoking we observed is especially encouraging, as the prevalence of smoking in the general population of a similar age to our patients has not materially changed in recent years. We do accept, however, that there may be some degree of selection bias in our study, as it was clinic based, and absconders (who may have an especially high and persistent smoking rate) were necessarily excluded. This potential bias, however, is likely to have operated identically in both the 1990 and 1999 survey, suggesting that the observed smoking reduction was real.

Our results thus give some cause for optimism, though with a 30% current smoking rate there is certainly no room for complacency. The use of nicotine replacement therapy, though beginning to be used now (see table 1), needs to be far more widely available, as it may significantly aid smoking cessation.


---

1st Asia Pacific Forum on Quality Improvement in Health Care

Three day conference

Wednesday 19 to Friday 21 September 2001

Sydney, Australia

We are delighted to announce this forthcoming conference in Sydney. Authors are invited to submit papers (call for papers closes on Friday 6 April), and delegate enquiries are welcome.

The themes of the Forum are:

- Improving patient safety
- Leadership for improvement
- Consumers driving change
- Building capacity for change: measurement, education and human resources
- The context: incentives and barriers for change
- Improving health systems
- The evidence and scientific basis for quality improvement.

Presented to you by the BMJ Publishing Group (London, UK) and Institute for Healthcare Improvement (Boston, USA), with the support of the Commonwealth Department of Health and Aged Care (Australia), Safety and Quality Council (Australia), NSW Health (Australia), and Ministry of Health (New Zealand).

For more information contact: quality@bma.org.uk or fax +44 (0)20 7383 6869