Survival in Patients With Type 2 Diabetes in a Swedish Community

Skaraborg Hypertension and Diabetes Project

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OBJECTIVE — To explore risk factors for all-cause mortality in patients with type 2 diabetes treated in primary care.

RESEARCH DESIGN AND METHODS — A prospective population-based study of 400 patients with type 2 diabetes who consecutively completed an annual checkup in primary care in Skara, Sweden, during 1992–1993. Vital status was ascertained to year 2000. Baseline characteristics as predictors for mortality were analyzed by Cox regression and expressed as relative risks (RRs), with 95% CIs.

RESULTS — During a mean follow-up time of 5.9 years, 131 patients died (56 deaths per 1,000 patients per year). In both sexes, all-cause mortality was predicted by HbA1c (by 1%; RR 1.14, 95% CI 1.01–1.27), and by LDL-to-HDL cholesterol ratios (1.15, 1.00–1.32). Increased mortality was also seen with prevalent hypertension (1.72, 1.21–2.44), microalbuminuria (1.87, 1.14, 95% CI 1.01–1.27), and previous cardiovascular disease (1.70, 1.15–2.50). Subanalyses revealed that increased mortality related to HbA1c was restricted to hypertensive patients with type 2 diabetes (1.23, 1.04–1.47). Serum triglycerides (by 1 mmol/l) predicted all-cause mortality in women (1.25, 1.06–1.47).

CONCLUSIONS — Poor glucose and lipid control and hypertension predicted all-cause mortality. Survival was also predicted by prevalent microalbuminuria and by previous cardiovascular disease. Confirming results from clinical trials, this population-based study has implications for primary and secondary prevention.

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Mortality in patients with diabetes is about four times higher than in nondiabetic subjects (1,2). In a large clinical trial (the U.K. Prospective Diabetes Study [UKPDS]), mortality did not differ significantly between those with intensive treatment of blood glucose and those with conventional treatment (3). However, in a subanalysis in overweight patients, a significant difference was found favoring intensive treatment of hyperglycemia (4). It was also demonstrated that intensive lowering of blood pressure was beneficial compared with less intensive lowering of blood pressure (5). Similar results have been reported from subanalyses in other randomized clinical trials on hypertension (6–8). From observational analyses, it has been shown that intensive blood glucose control is associated with a lower risk of any diabetes-related end point, diabetes-related death, and all-cause mortality in patients with type 2 diabetes (9,10). Furthermore, strong predictors for all-cause mortality, excluding increased blood glucose levels and hypertension, include lipoprotein abnormalities, high levels of serum triglycerides, and microalbuminuria (10–14).

In Sweden, the annual incidence of type 2 diabetes is ~16.1 per 100,000 inhabitants, and mortality in patients with diabetes is almost four times higher than in the general population (2). The objective of this population-based prospective study was to explore predictors for all-cause mortality identified in the UKPDS in patients with type 2 diabetes subjected to a structured diabetes education program in primary care in a Swedish community.

RESEARCH DESIGN AND METHODS

Skaraborg Hypertension and Diabetes Project

Since the 1970s, structured treatment and education programs for patients with hypertension and type 2 diabetes, respectively, have been used at the Primary Health Care Center in the municipality of Skara, Sweden. Annual check-ups of these patients have been performed (15–19), and the information has been computerized according to structured forms. In 1986, the hypertension and diabetes outpatient clinics in Skara merged, making a joint clinic with nurses educated on both diseases, supervised by the family physicians.

Subjects

Skara Health Care Center is the only available primary health care facility in the community and serves a total population of ~19,000 residents. Patients with type 2 diabetes who completed an annual check-up at the hypertension and diabetes outpatient clinic in Skara from June 1992 through September 1993 were eligible for the present study, and every patient seen gave informed consent to
participate in this study. The study enrolled 433 patients with diabetes. After exclusion of 33 patients with type 1 diabetes according to clinical criteria, 400 patients with type 2 diabetes (202 men and 198 women) remained for further analysis (20).

At the time of the study surveillance, 83% of the patients with diabetes residing in Skara who were found in the Skaraborg Diabetes Register (21) were reported from the Skara Health Care Center (B. Berger, personal communication). Of the 17% (n = 128) reported from other clinics in Skaraborg County, 40% were categorized as having type 1 diabetes and were thus not the target of our study. The remaining patients would mainly comprise patients with severe complications requiring specialist care at hospital clinics or with preferences for other clinics for other reasons. Accordingly, we assumed that the current study population, with few exceptions, would include nearly all of the patients with type 2 diabetes in Skara.

**Methods**

Nurses at the hypertension and diabetes outpatient clinic, who were specially trained for this task, carried out the examinations. The procedure has been described in detail previously (15). Information on medical history at baseline included information about when the patient with type 2 diabetes was first diagnosed. Blood specimens were drawn in the morning after a 10-h overnight fast. Routine tests, including fasting blood glucose and HbA1c (normal range 3.7–5.5%), were analyzed at the local hospital laboratory (Karnsjukhuset, Skövde, Sweden). HbA1c was measured by ion exchange high-performance liquid chromatography Mono S column (22). Serum samples for other tests were immediately frozen at -80°C and later analyzed for lipids (Lipids Laboratory, Lund University Hospital, Lund, Sweden) and serum insulin using a radioimmunoassay with <0.3% cross-reactivity for proinsulin (23) (kit from Pharmacia, Uppsala, Sweden; tests performed at the Wallenberg Laboratory, Malmö University Hospital, Malmö, Sweden). Height (to the nearest centimeter) and weight (to the nearest 0.1 kg) were measured (light indoor clothes and no shoes). BMI was calculated by dividing weight by height squared (kg/m²), and the waist-to-hip ratio (WHR) was calculated by dividing waist circumference (cm) by hip circumference (cm).

Diagnostic criteria for hypertension followed contemporary national guidelines. According to guidelines from 1987 up to the time of the present study, the definition was based on either ongoing treatment for hypertension or at least three consecutive readings of diastolic blood pressure ≥90 mmHg, irrespective of systolic blood pressure, in individuals older than 20 years of age (24). The treatment goal was set at diastolic blood pressure ≤90 mmHg.

Presence of microalbuminuria in urine was ascertained using a dipstick (Micral-Test) (25). Microalbuminuria was defined as ≥20 μg/l in the first morning sample of urine. A structured interview performed by the nurses included questions about medical history and current medications. The participants completed a detailed questionnaire about smoking habits, current alcohol consumption, and physical exercise in leisure time. The vital status of the cohort was ascertained through 31 December 1999 by record linkage with the Cause of Death Register at the National Board of Health and Welfare, Stockholm, Sweden.

Survival rate was modeled using Cox's proportional hazard model. With a level of significance at 0.05, there was an 80% power to detect a relative risk (RR) of 2.0 for all-cause mortality associated with a risk factor with a prevalence of 25%, given the rate of all-cause mortality during follow-up in this study population. To account for baseline imbalances in age and sex, these factors were included as covariates in all models; the results are thus presented in terms of age- and sex-adjusted RRs (RRadj) with 95% CIs. The RRadj for fasting blood glucose, total cholesterol, LDL cholesterol, and serum triglycerides relates to the marginal effect on survival of 1 mmol/l. The corresponding unit is 1% for HbA1c, 10 mmHg for systolic blood pressure, 5 mmHg for diastolic blood pressure, 5 years for duration of type 2 diabetes, 1 kg/m² for BMI, and 1 SD for fasting serum insulin, HDL cholesterol, and WHR. Because of skewed distributions, serum insulin and serum triglycerides were log-transformed in analyses and retransformed for tabulations. Cross-product interaction terms were used when further exploring the interactive aspects of significant findings in different subsamples in the study population.

The study protocol was approved by the Research Ethics Committee of the Medical Faculty, Göteborg University.

**RESULTS** — A total of 400 consecutively examined patients with type 2 diabetes (202 men and 198 women) were included in the present study. The mean follow-up time was 5.9 years, and 131 patients (67 men and 64 women) died during the observation time. The annual mortality rate was 56 deaths per 1,000 patients. Baseline characteristics and risk estimates for all-cause mortality are presented in Table 1. All-cause mortality was predicted by HbA1c, serum triglycerides, LDL-to-HDL cholesterol ratios, and co-morbidity presented as hypertension, previous cardiovascular disease, and microalbuminuria. An increased risk associated with current smoking was borderline significant (RRadj 1.66, 95% CI 0.99–2.76, P = 0.052). When stratified for sex, a similar pattern was found in both men and women.

In an analysis using all of the risk factors (except serum triglycerides) that were significant in Table 1, only previous cardiovascular disease (RRadj 1.67, 95% CI 1.04–2.68, P = 0.034) and microalbuminuria (1.88, 1.23–2.87, P = 0.003) were significant predictors for all-cause mortality, and hypertension (1.53, 0.997–2.34, P = 0.052) was a borderline significant predictor. When LDL-to-HDL cholesterol ratios were substituted by serum triglycerides in a corresponding analysis, only microalbuminuria (1.90, 1.26–2.87, P = 0.002) remained significant.

Survival curves of patients with type 2 diabetes stratified by the presence of hypertension and microalbuminuria without adjustment, respectively, are shown in Figs. 1 and 2. Hypertension (RRadj 1.81, 95% CI 1.23–2.67) and microalbuminuria (1.85, 1.21–2.83) were statistically significant predictors for mortality when also adjusting for differences in HbA1c, triglycerides, and LDL-to-HDL cholesterol ratios in addition to age and sex.

When comparing women with men and adjusting for differences in age, overall mortality risk was similar (RR 0.80, 95% CI 0.56–1.13). Age (1.57, 1.41–1.75, RR by 5 years) was a strong predictor for mortality but not diabetes duration (1.08, 0.95–1.23, RR by 5 years), with
RRs adjusted for differences in sex. When stratified for sex and adjusted for age, hypertension (1.99, 1.20–3.31), microalbuminuria (2.85, 1.67–4.87), and previous cardiovascular disease (1.79, 1.03–3.10) predicted mortality in men. In women, mortality was predicted only by fasting serum triglycerides (1.25, 1.06–1.47).

In hypertensive patients (both sexes combined) with type 2 diabetes, HbA1c (RRadj 1.23, 95% CI 1.04–1.47) and microalbuminuria (2.00, 1.20–3.31) were associated with increased mortality. All significant findings between sexes and the occurrence of hypertension were explored with analyses that used cross-product interaction terms. We were then able to confirm a sex-specific interaction in association with microalbuminuria (P = 0.02), but no other interaction terms were significant.

**CONCLUSIONS** — The main finding in this population-based prospective study was that survival in patients with type 2 diabetes is inversely related to poor glucose and lipid metabolism as well as to prevalent hypertension, microalbuminuria, and previous cardiovascular disease. Thus, the findings in the UKPDS (14) were confirmed in this community-based sample of patients with type 2 diabetes.

Markers for insulin resistance, such as overall (BMI) and central (WHR) obesity and fasting serum insulin, were not associated with increased mortality in this population. This is in accordance with previous results from the UKPDS showing that these factors did not predict coronary heart disease (14). In the general population, both overall and central obesity confer an increased risk of cardiovascular disease (26). However, results on the association between serum insulin and mortality are conflicting (27,28). Insulin resistance (29–31) and obesity (32–

**Table 1** — Baseline characteristics and corresponding RRs for all-cause mortality through 31 December 1999 in 400 subjects with type 2 diabetes in the Skaraborg Hypertension and Diabetes Project, 1992–1993

<table>
<thead>
<tr>
<th>Variables</th>
<th>Baseline characteristics</th>
<th>RR</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presence of hypertension</td>
<td>204 (51)</td>
<td>1.72</td>
<td>1.21–2.44</td>
<td>0.003</td>
</tr>
<tr>
<td>Previous cardiovascular disease</td>
<td>81 (20)</td>
<td>1.70</td>
<td>1.15–2.50</td>
<td>0.007</td>
</tr>
<tr>
<td>Presence of microalbuminuria</td>
<td>111 (28)</td>
<td>1.87</td>
<td>1.27–2.76</td>
<td>0.002</td>
</tr>
<tr>
<td>Current smoking</td>
<td>66 (17)</td>
<td>1.66</td>
<td>0.99–2.76</td>
<td>0.052</td>
</tr>
<tr>
<td>HbA1c (by 1%)</td>
<td>6.6 ± 1.5</td>
<td>1.14</td>
<td>1.01–1.27</td>
<td>0.031</td>
</tr>
<tr>
<td>Fasting blood glucose (by 1 mmol/l)</td>
<td>8.5 ± 2.5</td>
<td>1.05</td>
<td>0.96–1.14</td>
<td>0.284</td>
</tr>
<tr>
<td>Duration of type 2 diabetes (by 5 years)</td>
<td>8.3 ± 6.7</td>
<td>1.08</td>
<td>0.95–1.23</td>
<td>0.221</td>
</tr>
<tr>
<td>Systolic blood pressure (by 10 mmHg)</td>
<td>160 ± 22</td>
<td>1.06</td>
<td>0.98–1.15</td>
<td>0.150</td>
</tr>
<tr>
<td>Diastolic blood pressure (by 5 mmHg)</td>
<td>84 ± 10</td>
<td>1.03</td>
<td>0.94–1.12</td>
<td>0.548</td>
</tr>
<tr>
<td>BMI (by 1 kg/m²)</td>
<td>28 ± 4.6</td>
<td>0.99</td>
<td>0.95–1.03</td>
<td>0.654</td>
</tr>
<tr>
<td>WHR (by 1 SD)</td>
<td>0.92 ± 0.08</td>
<td>1.07</td>
<td>0.88–1.30</td>
<td>0.479</td>
</tr>
<tr>
<td>Fasting serum insulin (by 1 SD) (mU/l)*</td>
<td>22 ± 88</td>
<td>1.02</td>
<td>0.83–1.96</td>
<td>0.961</td>
</tr>
<tr>
<td>Total cholesterol (by 1 mmol/l)</td>
<td>5.9 ± 1.1</td>
<td>1.11</td>
<td>0.94–1.30</td>
<td>0.208</td>
</tr>
<tr>
<td>LDL cholesterol (by 1 mmol/l)</td>
<td>4.1 ± 1.0</td>
<td>1.12</td>
<td>0.93–1.34</td>
<td>0.226</td>
</tr>
<tr>
<td>HDL cholesterol (by −1 SD)</td>
<td>1.0 ± 0.2</td>
<td>1.15</td>
<td>0.98–1.35</td>
<td>0.084</td>
</tr>
<tr>
<td>LDL-to-HDL ratio (by 1 step)</td>
<td>4.2 ± 1.3</td>
<td>1.15</td>
<td>1.00–1.32</td>
<td>0.048</td>
</tr>
<tr>
<td>Triglycerides (by 1 mmol/l)*</td>
<td>1.8 ± 1.1</td>
<td>1.18</td>
<td>1.03–1.36</td>
<td>0.045</td>
</tr>
</tbody>
</table>

Data are n (%) or means ± SD. RR and P values for all-cause mortality were analyzed by Cox regression adjusted for age and sex. *Fasting serum insulin and triglycerides were log-transformed before analysis.

**Figure 1** — Survival curves of patients with type 2 diabetes with and without hypertension. The broken line represents patients without hypertension, and the filled line represents patients with hypertension. RR = 1.58 and 95% CI = 1.11–2.25, without adjustment, for patients with hypertension.
were seen per 1% difference in HbA1c. A recent prospective observational study from the UKPDS did show that each percent reduction in mean HbA1c was associated with a 14% reduction in all-cause mortality (9). In our study, we were also able to confirm this finding within the full sample by using cross-product interaction terms. However, cross-product interaction was found in association with microalbuminuria using a cross-product interaction term. However, cross-product interaction terms including sex and other risk factors were not significant, and consequently no other difference between the sexes could be confirmed. From these data, it is hard to conclude whether this could be explained by an inadequate number of patients (because the evaluation of cross-product interaction terms re-

Figure 2—Survival curves of patients with type 2 diabetes with and without microalbuminuria. The broken line represents patients without microalbuminuria, and the filled line represents patients with microalbuminuria. RR = 1.86 and 95% CI = 1.26–2.74, without adjustment, for patients with microalbuminuria.

34) are important in the development of type 2 diabetes and impaired glucose tolerance (35,36). However, once diabetes is established, these factors do not seem to be associated with higher mortality; instead, other risk factors become predominant determinants for all-cause mortality.

The results from our community-based cohort of patients with type 2 diabetes are consistent with previous clinical trials and observational studies reporting mortality in patients with type 2 diabetes. Predictors generally included high LDL cholesterol, low HDL cholesterol, high levels of serum triglycerides, high blood pressure, and high levels of HbA1c (5–14). A recent prospective observational analysis from the UKPDS did show that each percent reduction in mean HbA1c was associated with a 14% reduction in all-cause mortality (9). In our study, a corresponding 13% difference in survival was seen per 1% difference in HbA1c. A high prevalence of hypertension in patients with type 2 diabetes (15,37) and data showing hypertension as a risk factor for mortality in these patients have previously been reported in Skaraborg and other populations (5,38).

In a multivariate analysis including all risk factors associated with mortality, the strongest impact came from previous cardiovascular disease, prevalent microalbuminuria, and hypertension, confirming the importance of these risk factors in patients with type 2 diabetes.

The current antidiabetic treatment at baseline in the study population has been described in detail before (20), with the most frequent treatments being recommendations on diet (42%) and treatment with sulfonylureas (31%), metformin (0.5%), and insulin (11%). All patients categorized as hypertensive did receive pharmacological treatment, and the most frequently used antihypertensive drugs were β-blockers and diuretics. A possible protective effect from the use of β-blockers would, however, tend to underestimate the difference found in survival. We were not able to account for treatment modifications that took place after the baseline survey.

The finding of smoking being a statistically weak predictor for mortality may be due to an inadequate number of patients or to misclassification of smoking habits because of patients changing their behaviors after the diagnosis of diabetes. However, previous smokers might still have suffered from a remaining negative effect related to smoking.

No sex difference in survival was found in patients with type 2 diabetes, in accordance with previous reports (2,11). Indeed, type 2 diabetes appears to eliminate the relative protection against coronary heart disease and death seen in women without diabetes (39). We found increased levels of serum triglycerides to be a risk factor for mortality in women with type 2 diabetes, but not in men. This is in contrast to other prospective studies, where high serum triglycerides predicted mortality only in men (11) or overall (12).

It is plausible that increased serum triglycerides convey a higher mortality in diabetic women than in men, since increased triglycerides predict cardiovascular disease more consistently in women (40,41). One possible explanation is that women with type 2 diabetes respond differently to increased levels of serum triglycerides than do diabetic men. Alternatively, increased serum triglycerides in women with type 2 diabetes might express a more profound metabolic disturbance; the most commonly recognized risk factors of insulin resistance are highly correlated to each other (42). Furthermore, women have a more significant increase in triglyceride levels with the onset of diabetes (43). Microalbuminuria predicted mortality only in men, also in accordance with a previous report (11). In our study, we were also able to confirm this finding within the full sample by using cross-product interaction terms. However, the conclusions regarding microalbuminuria are limited by the fact that optimal ascertainment of microalbuminuria as a diagnosis requires timed overnight urine samples on two or three different days (25). However, even if our dichotomization of microalbuminuria as present or absent confers some misclassification in terms of standardized diagnosis, we still found significant differences associated with survival.

Thus, the pattern of predictors for mortality in men was different from that seen in women. A true sex-specific interaction was found in association with microalbuminuria using a cross-product interaction term. However, cross-product interaction terms including sex and other risk factors were not significant, and consequently no other difference between the sexes could be confirmed. From these data, it is hard to conclude whether this could be explained by an inadequate number of patients (because the evaluation of cross-product interaction terms re-
reasures a higher power) or by a lack of a true difference.

A previous analysis of the same popu-
lation indicated that patients with both
type 2 diabetes and hypertension had
higher BMIs, higher triglycerides, higher
LDL-to-HDL cholesterol ratios, and
higher fasting serum insulin. Conversely,
glucose levels were lower than those in
normotensive patients with type 2 dia-
teses (20). The clustering of cardiovascular
risk factors has been found to elevate the
mortality risk profoundly (38). These ob-
servations contribute to the understand-
ing of the increased mortality associated
with the combined occurrence of type 2
diabetes and hypertension.

In the present study, the only predic-
tor for mortality in patients with both
type 2 diabetes and hypertension was HbA1c
level, despite their lower mean HbA1c lev-
els compared with those with type 2 dia-
tes alone. Moreover, in this population
it has previously been shown that patients
with both type 2 diabetes and hyperten-
sion have been associated with the combined occurrence of type 2
diabetes and hypertension.

In conclusion, risk factors for mortality in
this community-based cohort of pa-
tients with type 2 diabetes were poor
glycemic control, dyslipidemia, and hypo-
tension. Survival also differed in sub-
groups of comorbidity. Previous find-
ings in clinical trials and observational studies
were thus consistently confirmed in this
ethnically homogeneous primary care
population that included the vast major-
ity of people with type 2 diabetes in a
geographically defined area. Implications
for primary and secondary prevention are
evident.

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