

Risk Factors for Mortality in a Diverse Cohort of Patients With Childhood-Onset Diabetes in Chicago

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OBJECTIVE — We sought to determine whether risk of death differed by demographic or other characteristics in a population-based cohort of patients with childhood-onset diabetes.

RESEARCH DESIGN AND METHODS — The Chicago Childhood Diabetes Registry is an ethnically diverse cohort of patients with diabetes onset between ages 0 and 17 years. Cases that accumulated from 1 January 1985 to 31 December 2000 ($n = 1,238$) were assessed for vital status using death certificates, family reports, and two large administrative databases (Social Security Death Index and National Death Index). Mortality was compared between subgroups using Poisson and Cox proportional hazards regression.

RESULTS — Thirty subjects died, with a mean follow-up time of 7.75 years, yielding a crude case fatality rate of 2.4%. Six subjects died of diabetic ketoacidosis (DKA) at initial presentation. Onset age was the predominant risk factor, driven by a substantially higher fatality rate among those diagnosed at age 17 years (13.36/1,000 person-years) than among other ages and by moderately higher rates for those diagnosed in early adolescence (ages 10–13 years) (3.49/1,000 person-years), compared with children diagnosed before age 10 years (0.89/1,000 person-years) or at ages 14–16 years (0.81/1,000 person-years). Mortality did not differ significantly by other factors examined; data for whites were insufficient to allow comparisons with that group. In addition to diabetes, frequent causes of death were trauma, infectious disease, and cardiovascular disease.

CONCLUSIONS — This study provides short- to medium-term follow-up in a diverse cohort of patients. DKA remains a significant cause of death in young people with diabetes. Young people diagnosed at the threshold of adulthood are at increased risk for mortality.

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Despite advances in care, mortality due to childhood-onset diabetes remains significant. Long-term complications can be forestalled with intensive blood glucose control (1,2); however, optimal therapy may not be uniformly available. Population-based studies from Scandinavia (3–5), the U.K. (6,7), and Estonia and Lithuania (8) show increased mortality for patients with youth-onset diabetes compared with that

for the general population. Great disparities in mortality exist between countries (8–10). In the Diabetes Epidemiology Research International Mortality Study (11,12), northern and central Europe and Canada reported the best outcomes. Eastern Europe, Japan, and Russia had the highest mortality, with case fatality rates 5–10 times higher than in Norway, which was chosen as a standard. Mortality for youth-onset diabetes in Allegheny County,

Pennsylvania (9), was found to be 2.5 times higher than in Israel and 1.6 times higher than in Finland, suggesting that some proportion could be preventable. In Allegheny County (13), African Americans died more often from acute complications of diabetes than did whites; Roy et al. (14) also found increased mortality among African-American patients with type 1 diabetes in New Jersey. DiLiberti et al. (15) used national data to study temporal trends for childhood diabetes mortality. Death rates were significantly higher in areas with low socioeconomic status and somewhat higher in areas lacking sophisticated medical care. Among 15- to 19-year-old patients, diabetes mortality declined through 1977 and then increased. Socioeconomic and access factors may underlie the recent plateau of childhood diabetes mortality, despite ongoing technological advances.

Lipton et al. (16) studied racial differences in childhood diabetes mortality in Chicago, where case fatality rates were 9.3 times higher for African Americans than for whites. Acute complications accounted for 70% of African-American deaths from diabetes, 80% of Latino deaths, and one of the two deaths among whites, suggesting that socioeconomic and access disparities likely underlie the racial differential in mortality in Chicago. That study was limited by the small number of whites and cross-sectional rate comparison, using calculated population estimates rather than a longitudinal design.

The current population-based study followed a large, diverse cohort of patients with childhood-onset diabetes in Chicago to determine whether mortality risk differed by ethnicity, age at diagnosis, sex, or other characteristics in short- to medium-term follow-up.

RESEARCH DESIGN AND METHODS

The Chicago Childhood Diabetes Registry comprises a cohort of subjects diagnosed with diabetes before age 18 years and residing in Chicago at diagnosis. The primary source of ascertainment is hospital records, augmented by outpatient sources. Eligible cases are identified by medical records re-

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Abbreviations: DKA, diabetic ketoacidosis; NDI, National Death Index; SMR, standardized mortality ratio.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

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Table 1—Crude mortality rates (including those who died at onset)

Group	n	Deaths	Person-years	Rate/1,000 person-years (95% CI)	Rate ratio
All	1,238	30	9,596	3.13 (2.19–4.47)	
Female	680	14	5,200	2.69 (1.59–4.55)	1
Male	558	16	4,397	3.64 (2.23–5.94)	1.35
Onset age (years)					
0–9	433	5	3,370	1.48 (0.62–3.57)	1
10	75	2	617	3.24 (0.81–12.96)	2.18
11	101	3	722	4.15 (1.34–12.88)	2.80
12	108	2	824	2.43 (0.61–9.71)	1.64
13	129	5	990	5.05 (2.10–12.13)	3.40*
14	116	1	988	1.01 (0.14–7.19)	0.68
15	107	0	815	—	—
16	93	3	681	4.40 (1.42–13.65)	2.97
17	76	9	590	15.26 (7.94–29.32)	10.28†
Onset age (years)					
0–9	433	5	3,370	1.48 (0.62–3.56)	1
10–13	413	12	3,152	3.81 (2.16–6.70)	2.57*
14–16	316	4	2,484	1.61 (0.60–4.29)	1.09
17	76	9	590	15.26 (7.94–29.32)	10.28†
Latino	385	7	2,935	2.39 (1.14–5.00)	1
African American	853	23	6,662	3.45 (2.29–5.20)	1.45
Onset 1985–1989	350	11	4,636	2.37 (1.31–4.28)	1.06
Onset 1990–1994	436	16	3,611	4.43 (2.71–7.23)	1.99
Onset 1995–2000	452	3	1,350	2.22 (0.72–6.89)	1
Private insurance	317	7	2,220	3.15 (1.50–6.61)	1
Public/no health insurance	443	9	3,182	2.83 (1.47–5.44)	0.90
Health insurance unknown	472	8	4,194	1.91 (0.95–3.81)	0.60
Presumed type 1 diabetes	868	17	7,164	2.37 (1.48–3.82)	1
Presumed type 2 diabetes	364	7	2,432	2.88 (1.37–6.04)	1.21

*P < 0.10; †P < 0.001.

view at 37 of 40 area hospitals with a pediatric ward and at least 200 total inpatient beds; patients are included if they were diagnosed after 31 December 1984 and a resident of Chicago at the time of diagnosis. Before 1 January 1992, only children discharged on insulin treatment and classified as African American or Latino were registered. More recently, all young Chicago residents diagnosed with diabetes were included, irrespective of initial treatment or ethnic group. Ethnicity is defined as non-Latino white, non-Latino African American, or Latino by the medical record and/or having a surname classified as Hispanic by the U.S. Census Bureau (17). Those with Latino surnames but medical record classification as non-Latino white or non-Latino African American were considered Latino. American Diabetes Association camp lists from northern Illinois, a survey of unaffiliated neighborhood clinics, medical chart review at 16 clinics maintained by the Chicago Department of Health, and the

Illinois Department of Public Aid payment database served as secondary sources of cases. Overall completeness of ascertainment was estimated at 86% for the years 1985–1990 and 84% for 1991–2000, using the capture-mark-recapture method (18). The University of Chicago institutional review board and those at other participating institutions approved this study.

Vital status was determined for the cohort of African American and Latino cases identified from 1 January 1985 to 31 December 2000 (n = 1,238), using the National Death Index (NDI), Social Security Death Index, and individual case follow-up. The NDI is a registry compiled from local vital statistics records nationwide. Subjects' names, addresses, birth dates, and social security numbers (when known) were matched to NDI records. Subjects were considered exact matches if name, birth date, and social security number all matched; cases missing one or two items were considered likely matches and

explored on a case-by-case basis. The Social Security Death Index, a large publicly available database, was also searched online and matched to the Chicago database. Death certificates and reports of death by immediate family or subjects' primary care providers were also considered definitive evidence of mortality. Records were searched through 31 December 2000; after that date, unmatched cases were considered to have survived and were censored.

We distinguished a group of probable type 2 diabetic patients within the cohort based on documentation in the medical record of one or more of the following: obesity, BMI at onset \geq 95th percentile for age and sex (19), physician note of "possible type 2" or "atypical" diabetes, acanthosis nigricans, polycystic ovary syndrome, or treatment with oral hypoglycemics. In addition, those responding positively to specific questions during interviews were considered likely to have type 2 diabetes, including cessation of insulin after the "honeymoon" period (for >2 weeks and >6 months after initial diagnosis) or current treatment with oral hypoglycemics. Those not meeting any of these criteria were considered to have type 1 diabetes.

Person-years of follow-up were calculated from the date of diagnosis through the date of death or 31 December 2000. For one individual reported to have died but not appearing in the NDI, we considered the date reported by the health care provider the date of death. Poisson and Cox proportional hazards regression analysis were used to test univariate and multivariate predictors of mortality.

RESULTS— This cohort provided 9,596 person-years of follow-up: on average, 7.62 years in Latinos and 7.81 years in non-Latino African Americans (Table 1). Among the 1,238 cases of youth-onset diabetes with a mean follow-up of 7.75 years, 30 subjects were found to have died, for an overall case fatality rate of 2.42%. Seven deaths occurred among Latinos, and 23 deaths occurred among non-Latino African Americans. Six patients died at onset (before or within initial hospitalization for new-onset diabetes). Of these six, four were African American and two were Latino. Those dying at onset were excluded from the analysis of risk factors for mortality after initial stabilization. No deaths occurred among non-Latino whites and others in the Registry database, so they were excluded from this

Table 2—Crude mortality rates (excluding those who died at onset)

Group	n	Deaths	Person-years	Rate/1,000 person-years (95% CI)	Rate ratio
All	1,232	24	9,596	2.50 (1.68–3.73)	
Female	677	11	5,200	2.12 (1.17–3.82)	1
Male	555	13	4,397	2.96 (1.72–5.09)	1.40
Onset age (years)*					
0–9	431	3	3,370	0.89 (0.29–2.76)	1
10	75	2	617	3.24 (0.81–12.96)	3.64
11	101	3	722	4.15 (1.34–12.88)	4.67†
12	108	2	824	2.43 (0.61–9.71)	2.73
13	128	4	990	4.04 (1.52–10.76)	4.54‡
14	116	1	988	1.01 (0.14–7.19)	1.13
15	107	0	815	—	—
16	91	1	681	1.47 (0.21–10.42)	1.65
17	75	8	590	13.36 (6.78–27.12)	15.24§
Onset age (years)					
0–9	431	3	3,370	0.89 (0.29–2.76)	1
10–13	412	11	3,152	3.49 (1.93–6.30)	3.92‡
14–16	314	2	2,483	0.81 (0.20–3.22)	0.90
17	75	8	590	13.56 (6.78–27.12)	15.24§
Latino	383	5	2,935	1.70 (0.71–4.09)	1
African American	849	19	6,662	2.85 (1.82–4.47)	1.67
Onset 1985–1989	349	10	4,636	2.16 (1.16–4.01)	1
Onset 1990–1994	431	11	3,611	3.05 (1.69–5.50)	1.41
Onset 1995–2000	452	3	1,350	2.22 (0.72–6.89)	1.03
Private insurance	317	7	2,220	3.15 (1.50–6.61)	1
Public/no health insurance	443	9	3,182	2.83 (1.47–5.44)	0.90
Health insurance unknown	472	8	4,194	1.91 (0.95–3.81)	0.60
Presumed type 1 diabetes	868	17	7,164	2.37 (1.48–3.82)	1
Presumed type 2 diabetes	364	7	2,432	2.88 (1.37–6.04)	1.21

*When ages 14–15 years were combined, rate/1,000 person-years = 0.55 (95% CI 0.08–3.94) and rate ratio = 0.62. †P < 0.10; ‡P < 0.05; §P < 0.001.

analysis because of their relatively short length of follow-up (4.29 years) over a time period that began 7 years later than for the minority youth reported here.

Among those surviving their initial diagnosis, mean \pm SD duration was 7.79 ± 4.47 years, ranging from 1 day to 15.97 years (median 7.70 years) (Table 2). The highest mortality rate was observed among subjects diagnosed at age 17 years (13.36/1,000 person-years). In contrast, mortality for youth diagnosed at ages up to 16 years ranged from 0.89 to 4.15/1,000 person-years. Higher mortality occurred among those diagnosed in early adolescence (ages 10–13 years) (3.49/1,000 person-years) than in children diagnosed before age 10 years (0.89/1,000 person-years) or in their mid-teens (ages 14–16 years) (0.81/1,000 person-years).

Figure 1 represents survival by age at onset. We found no significant differences in survival by sex, ethnicity, period of diagnosis (1985–1989, 1990–1994,

and 1995–2000), diagnosis in a tertiary care facility versus community hospital,

insurance status at onset, or presumed phenotype (Table 3).

Overall mortality was significantly higher in this cohort of young people with diabetes than in the comparable age-matched population in Chicago. Standardized mortality ratios (SMRs) were calculated for the overall cohort and by ethnicity, since African-American youth in Chicago are at approximately three times the risk for death as Latinos and non-Latino whites. Mortality rates for Chicago residents aged 5–29 years from 1989 to 2000 averaged 0.61/1,000 person-years among whites, 1.82/1,000 person-years among African Americans, and 0.71/1,000 person-years among Latinos (Chicago Department of Health, unpublished data), yielding an SMR of 1.90 (95% CI 1.14–2.96) for young African Americans with diabetes and 3.37 (1.35–6.94) for young Latinos.

Examination of causes of death (Table 4) revealed DKA as the number one cause, even among those initially stabilized. “Diabetes” alone was listed on one death certificate, probably due to acute complications. Cardiovascular disease was relatively frequent: three patients in their 20s died of stroke or heart disease, while a fourth dying of DKA was found to have autopsy evidence of coronary artery disease. None of the decedents had diabetic nephropathy listed on their death certificates. Five trauma deaths occurred (one death certificate simply listed “cause under police investigation”). Infection ($n = 2$) and other causes ($n = 3$) accounted for the remainder of deaths.

Kaplan-Meier Survival Estimates by Age at Onset

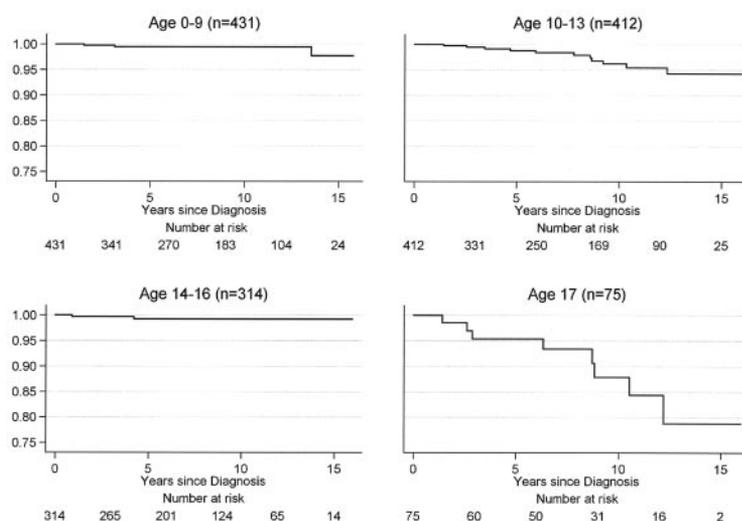


Figure 1—Kaplan-Meier survival estimates by age at onset. Number at risk at the beginning of each time interval are shown on plots.

Table 3—Cox regression analysis of time to death versus patient characteristics

Group	n*	Deaths*	Hazard ratio (95% CI)	P†
Female	667	11	1.00	—
Male	555	13	1.38 (0.62–3.09)	0.43
Onset age (years)*				
0–9	431	3	1.00	—
10	75	2	3.53 (0.59–21.11)	0.17
11	101	3	4.81 (0.97–23.83)	0.055
12	108	2	2.72 (0.45–16.26)	0.27
13	128	4	4.64 (1.04–20.75)	0.04
14	116	1	1.16 (0.12–11.11)	0.90
15	107	0	—	—
16	91	1	1.70 (0.18–16.26)	0.65
17	75	8	15.21 (4.04–57.36)	<0.001
Onset age (years)				
0–9	431	3	1.00	—
10–13	412	11	3.94 (1.09–14.14)	0.04
14–16	314	2	0.93 (0.16–5.56)	0.94
17	75	8	15.21 (4.03–57.3)	<0.001
Latino	383	5	1.00	—
African American	849	19	1.67 (0.62–4.47)	0.31
Onset 1985–1989	349	10	1.00	—
Onset 1990–1994	431	11	2.15 (0.79–5.85)	0.13
Onset 1995–2000	452	3	1.94 (0.42–8.93)	0.40
Private insurance	317	7	1.00	—
Public/no health insurance	443	9	0.90 (0.33–2.41)	0.83
Health insurance unknown	472	8	0.57 (0.20–1.56)	0.27
Presumed type 1 diabetes	868	17	1.00	—
Presumed type 2 diabetes	364	7	1.29 (0.53–3.13)	0.57

*Excluding those who died at onset. †Overall significance levels for categorical factors: onset age (9-level) 0.0012, onset age (4-level) <0.0001, onset period 0.38, diagnosis location 0.32, and insurance status 0.48.

CONCLUSIONS— Across countries and within the U.S., mortality experience for patients with childhood-onset diabetes varies by the overall health status of the underlying population, socioeconomic status, and access to high-quality medical care and ongoing diabetes management. Despite advanced technology, many apparently preventable deaths occur. Vanelli et al. (20) demonstrated a dramatic reduction in DKA through a regional education program in Italy. Targeted efforts may improve outcomes for regions with less experience (as demonstrated in Japan) and populations with increased rates of acute complications (African Americans and Latinos in the U.S.). This study, in a cohort of minority subjects with childhood-onset diabetes in Chicago, identified increased mortality risk among subjects with postpubertal onset. Previous studies of mortality from childhood-onset diabetes have not distinguished cases with postpubertal onset (3–16) or type 1 versus type 2 diabetes or have only studied patients presumed to have type 1 diabetes (3,5,6,8,10,14). Au-

thors of one study comparing international death rates asserted that the bulk of the excess mortality should be attributed to type 1 diabetes (9). Recently, Liang et al. (7) found that “psychosocial factors are powerful risk factors for mortality from acute events in patients with type 1 diabetes.” Our findings are consistent with those of other investigators in the U.K.

Table 4—Causes of death

Cause of death	n	Age at death	Pubertal onset	African American	Male
Acute diabetes complications					
Died at onset	6	(9.2–17.7)	67 (4)	67 (4)	50 (3)
DKA after onset*	7	(11.3–26.3)	57 (4)	71 (5)	43 (3)
Cardiovascular disease	4	(24.1–27.9)	100 (4)	100 (4)	25 (1)
Infection	2	(10.7–18.8)	50 (1)	100 (2)	50 (1)
Trauma	5	(15.7–24.1)	80 (4)	80 (4)	100 (4)
Other causes†	3	(13.3–30.2)	67 (2)	67 (2)	33 (1)
Unknown causes‡	3	(17.1–23.0)	67 (2)	67 (2)	67 (2)

Data are ranges or % (n) unless otherwise indicated. *Includes one death simply documented as “diabetes” on death certificate. †Crohn’s disease, Hunter’s syndrome, and pulmonary embolism. ‡Two of these death dates were estimated by family or caregiver.

and northern Europe who found a significant proportion of mortality due to violence and acute complications in young people with diabetes (7,8). The lack of observed mortality related to nephropathy in the Chicago cohort may be due in part to the sample size and to their youth and relatively short duration of diabetes. Similar logic may explain the comparatively low number of cardiovascular deaths in this sample. The high proportion of deaths from DKA could reflect deficiencies in recognition of warning signs, access to care, or quality of care for DKA during this time period in the sample studied. The lack of deaths among those aged 0–4 years at diagnosis is most likely due to the small number of cases in this age-group. The high risk of death for patients diagnosed during adolescence could reflect that these patients are essentially out of their parents’ control at the time of diagnosis, increasing the risk of DKA and/or poor overall diabetes management.

Limitations of this study include the relatively low overall number of deaths, affording low statistical power for most subgroup comparisons. Whites and others were added to the database only after 1992, limiting the numbers and person-years available for comparison, so they were excluded from this analysis. Subsequent analyses will include whites and other ethnicities, as years of observation accumulate. Obesity alone may not be a reliable indicator of type 2 diabetes. Our case ascertainment rate of 85% means that some patients were not captured in this dataset, including some who may have died at onset. We did not examine postmortem records, which might have clarified the circumstances of these deaths. Death certificate data are not always specific; it is possible that some of

the deaths recorded as “trauma” were actually cases of suicide. The increased death rate among patients with onset at age 17 years may be an artifact of less complete ascertainment of “mild” cases of diabetes in older adolescents treated as outpatients and, therefore, an artificially small denominator. As further data accumulate, additional patterns may emerge.

Physicians should be aware of the increased risk of death in children diagnosed with diabetes during or after puberty. Targeting educational, outreach, and prevention efforts among those who provide their health care is warranted. Moreover, death is simply the most severe diabetes outcome. Complication rates for youth-onset type 2 diabetes are not yet well documented (21), although some investigators believe that youth-onset type 2 diabetes may exhibit a more aggressive course than adult-onset disease (22). Among adults, minority patients with diabetes experience increased complication rates (23). When minority youth develop diabetes, they may also be at increased risk for early morbidity and disability, in addition to the risk of mortality. Broad public health and policy measures are needed to reduce excess morbidity and mortality from diabetes, especially among patients with pubertal onset of disease.

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