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Impact of the era of diabetes onset in a national care system on the prevalence of retinopathy and microalbuminuria in people living with type 1 diabetes 15 years post-diagnosis: A cross-sectional, real-world observational study

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Abstract

Aims: This study investigates the impact of the era of diabetes onset on the prevalence of diabetic retinopathy and albuminuria 15 years post-diagnosis in people living with type 1 diabetes (T1D) within a national healthcare system offering structured multidisciplinary endocrinology care.

Materials and Methods: We analysed data of 2176 individuals diagnosed with T1D before age 30, comparing two cohorts based on diabetes onset period: group A (1985–1998) and group B (1998–2009). The prevalence of diabetic retinopathy and albuminuria was assessed using generalised estimating equations.

Results: Glycaemic control (haemoglobin A1C: 8.3% vs. 8.1%, $p < 0.0001$) and low density lipoprotein cholesterol (2.6 mmol/L vs. 2.5 mmol/L, $p < 0.001$) were poorer in group A, whereas obesity prevalence was higher in group B (12.8% vs. 17.4%, $p < 0.01$). Group A used more antihypertensive therapy (14.9% vs. 11.5%, $p < 0.01$), while group B used more lipid-lowering therapy (9.2% vs. 15.9%, $p < 0.0001$). The prevalence of diabetic retinopathy 15 years post-diagnosis significantly declined from 36.3% in 2001 to 21.1% in 2022 ($p < 0.0001$). This decrease was particularly pronounced in individuals with diabetes onset after 11 years of age. The prevalence of albuminuria, adjusted for age and sex, decreased from 14.9% in 2001 to 7.3% in 2022 ($p < 0.05$).

Conclusions: Individuals diagnosed with T1D after 1998 had fewer microvascular complications 15 years post-diagnosis, especially less retinopathy in those with diabetes onset after age 11. This decline highlights the impact of improved care.

KEYWORDS
microvascular complications, real-world data, type 1 diabetes

1 | INTRODUCTION

Over the past decades, type 1 diabetes (T1D) care has significantly improved, driven by advancements in both glucose management and overall metabolic control, including blood pressure and lipid management. Innovations such as novel cardiovascular medications, new insulin analogues, advanced insulin pump systems and glucose monitoring technologies have played a crucial role in these improvements. These improvements are reflected in a decline in diabetes-related micro- and macrovascular complications and reduced mortality rates among individuals with T1D.[1–5] However, much of this evidence is derived from clinical studies; while population-level data remain scarce. In the wider T1D population, the uptake of advanced technologies and the effectiveness of diabetes management strategies vary considerably between countries and populations.[6,7]

In Belgium, people requiring intensive insulin therapy—whether through multiple daily injections (MDI) or continuous subcutaneous insulin infusion (CSII)—benefit from multidisciplinary specialist care through the Diabetes Convention system.[8] Under this Diabetes Convention, hospital-based diabetes centres provide free-of-charge multidisciplinary specialist care, including access to diabetes education, glucose monitoring materials like continuous glucose monitoring (CGM), CSII and hybrid closed-loop insulin delivery systems, irrespective of any glucose criteria. Regular follow-up by a team comprising an endocrinologist or paediatrician specialised in diabetology, diabetes nurses and educators, dietitians, podiatrists and psychologists is foreseen. Nearly all people with T1D participate in this programme, enjoying full reimbursement for insulin analogues, insulin pens, CSII and glucose monitoring tools and technologies. Since 2001, the Initiative for Quality Improvement and Epidemiology in Diabetes (IQED) has been routinely collecting real-world clinical data from adults participating in the Diabetes Convention to monitor patient characteristics and care quality.[8,9] Feedback of individual centre data compared to anonymised data of other centres (also known as benchmarking) is given to each centre to promote quality improvement measures.

The aim of this study was to investigate in a real-world setting the impact of the era of diabetes onset on the prevalence of complications 15 years post-diagnosis in people living with T1D followed in a health care system with full access to specialist-supported diabetes care and tools.

2 | MATERIALS AND METHODS

2.1 | Study population

The IQED database contains data on adults (aged ≥18 years, adjusted to ≥16 years from 2016 onward) diagnosed with type 1 or type 2 diabetes from 2001 to 2022 (= last data collection available at the moment of this study). Individuals with pancreas or islet cell transplantation, dementia or pregnancy were excluded from the IQED database. Additionally, between 2006 and 2014, data from individuals receiving CSII therapy were not collected in the IQED database. Participating centres conducted biennial reviews of patient records, completing a standardised electronic questionnaire with the most recent data from the previous year, representing an audit period. For every data collection, a random letter of the alphabet was picked by the central IQED team and communicated to the participating centres. Centres needed to extract an alphabetically ordered list of all patients followed in their centre and report data of these patients starting from the random letter, continuing until they reached 10% of their total patient population with a minimum of 50 patients. To ensure randomness and representativeness, the starting letter for selection was communicated only at the start of the data collection process. For optimal data validity, inconsistent data are reported back to participating centres, corrected if necessary and re-entered into the database. All data were pseudonymised. Detailed information on the parameters collected and the IQED data collection process is available online.[9]

2.2 | Final dataset and study groups

For this study, individuals with a clinical diagnosis of T1D, as recorded in electronic patient files and reported by the clinician, were selected ($N = 26\,847$). To further minimise the inclusion of individuals with potential misclassification as type 2 diabetes, only those with diabetes onset before the age of 30 years were included ($N = 15\,215$). As our focus was on the 15-year post-diagnosis stage, we included only data from that time point (range: 13–16 years post-diagnosis, given the biennial schedule of IQED assessments), yielding a final dataset of 2176 individuals. Where duplicate registrations existed within this timeframe, only the latest record was retained.

The progression of complications at 15 years post-diagnosis was examined over the total 20-year study period, starting in 2001 when a new system for quality control of diabetes care was launched in Belgium (IQED).[8] We have taken the available data collected over the next 20 years and have divided this period into two equal parts. Group A included 1188 individuals who were diagnosed with T1D between 1985 and 1998 (read-out years 2001–2011). Group B included 988 individuals who were diagnosed with T1D between 1998 and 2009 (read-out years 2012–2022).

2.3 | Parameters

Diabetes retinopathy was captured as 'Has the patient ever received treatment (laser photocoagulation and/or intravitreal injection) for diabetic retinopathy?' and 'Does the patient have diabetic retinopathy?' where yes = 1, no = 0, no answer = 9. As help text the following info was given: 'Also answer 'Yes' if the retinopathy is inactive. Also answer "Yes" if only 1 eye is affected. Also answer "Yes" if you have indicated that this patient has ever been treated (laser photocoagulation and/or intravitreal injection) for diabetic retinopathy' Albuminuria was assessed by a spot urine test or 24-h urine collection as albumin >0.03 g/L (>30 mg/L). The definition of diabetic retinopathy

and albuminuria were kept stable over the entire study period, limiting detection bias.

Age at diabetes onset was categorised into three groups: 0–11, 12–17 and 18–29 years.

The low density lipoprotein (LDL) cholesterol was calculated by the Friedewald formula for the people with triglycerides < 4.52 mmol/L (< 400 mg/dL) regardless of the condition of the blood sample (fasted and non-fasted).[10,11]

2.4 | Statistical analysis

Results are expressed as proportions for categorical variables or mean ± standard deviation (SD) for normally distributed variables. Pairwise differences of the population characteristics between group A and group B were analysed using generalised estimating equations (GEE); using logistic regression for dichotomous outcome variables and the normal probability distribution for continuous outcome

TABLE 1 Population characteristics by study group.

	Group A	Group B	*p*-Value
Study period	2001–2011	2012–2022	
Diabetes onset (from–to)	1985–1998	1998–2009	
N=	1188	988	
Sex (male)	684 (57.6)	570 (57.7)	NS
Age, years	32.7 ± 7.6	31.6 ± 7.8	<0.001
Diabetes duration, years	14.9 ± 1.2	15.2 ± 1.1	<0.0001
Age at diabetes onset, years	17.8 ± 7.6	16.4 ± 7.7	<0.0001
Age at diabetes onset, categories			
0–11 years	328 (27.6)	353 (35.7)	<0.0001
12–17 years	273 (22.3)	229 (23.2)	NS
18–29 years	587 (49.4)	406 (41.1)	<0.0001
BMI	(*N* = 1086)	(*N* = 922)	
BMI, kg/m^2	25.5 ± 4.5	26.1 ± 4.6	<0.01
BMI categories			
Normal weight, <25 kg/m^2	571 (52.6)	428 (46.4)	<0.01
Overweight, 25 to <30 kg/m^2	376 (34.6)	334 (36.2)	NS
Obesity, ≥30 kg/m^2	139 (12.8)	160 (17.4)	<0.01
Systolic BP, mmHg	124.1 ± 14.8 (*N* = 1150)	124.4 ± 15.0 (*N* = 961)	NS
Diastolic BP, mmHg	75.4 ± 9.6 (*N* = 1150)	75.7 ± 10.3 (*N* = 960)	NS
BP categories	(*N* = 1150)	(*N* = 960)	
<140/90 mmHg	910 (79.1)	778 (81.0)	NS
<130/85 mmHg	689 (59.9)	572 (59.6)	NS
Lipid profile	(*N* = 1079)	(*N* = 867)	
Total cholesterol, mmol/L	4.8 ± 0.9	4.6 ± 0.9	< 0.0001
Triglycerides, mmol/L	1.2 ± 0.7	1.2 ± 0.7	NS
HDL-cholesterol, mmol/L	1.6 ± 0.7	1.6 ± 0.5	<0.05
LDL-cholesterol, mmol/L	2.6 ± 0.8	2.5 ± 0.8	<0.001
LDL-cholesterol categories			
<100 mmol/L	568 (52.6)	495 (57.1)	NS
<70 mmol/L	146 (13.5)	160 (18.5)	<0.01
Smoking (yes)	249 (22.4) (*N* = 1110)	190 (20.6) (*N* = 924)	NS
Use of antihypertensive therapy[a]	172 (14.9) (*N* = 1155)	111 (11.5) (*N* = 968)	<0.01
Use of lipid-lowering therapy[b]	106 (9.2) (*N* = 1156)	153 (15.9) (*N* = 962)	<0.0001
Insulin pump use[c]	12 (1.0) (*N* = 1176)	134 (13.8) (*N* = 968)	<0.0001
CGM use[d]	ND	572 (58.8) (*N* = 972)	ND
Diabetes control	(*N* = 1170)	(*N* = 970)	
HbA1c, mmol/mol	67.4 ± 17.9	64.6 ± 15.9	<0.0001

(Continues)

TABLE 1 (Continued)

	Group A	Group B	*p*-Value
HbA1c, %	8.3 ± 1.6	8.1 ± 1.5	<0.0001
HbA1c categories			
<7.5%	363 (31.0)	380 (39.2)	<0.001
≥7.5 to <9%	476 (40.7)	367 (37.8)	NS
≥9%	331 (28.3)	223 (23.0)	<0.01

Note: Table 1 shows the comparison of study population characteristics 15 years post-diagnosis by study group. Values are *n* (%) for proportions or mean ± standard deviation for continuous variables. Data on BMI, blood pressure, lipid profile, smoking status, therapy and diabetes control were only available for the indicated populations (*N*).

Abbreviations: BMI, body mass index; BP, blood pressure; CGM, continuous glucose monitoring; HbA1c, haemoglobin A1C; HDL, high density lipoprotein; LDL, low density lipoprotein; *N*, population size; *n*, number of observations; ND, not determined; NS, not significant.

[a]Antihypertensive therapy has been defined as the use of either Renin-Angiotensin-Aldosterone System blockers (angiotensin-converting enzyme [ACE] inhibitors and sartans) or other antihypertensive drugs (included in the IQED data collection from 2006).

[b]Lipid-lowering therapy defined as the use of either one of these classes: statins, fibrates (included in the Initiative for Quality Improvement and Epidemiology in Diabetes (IQED) data collection from 2006), ezetimibe (included in the IQED data collection from 2011).

[c]Continuous subcutaneous insulin infusion users were not eligible for inclusion in the IQED study between 2006 and 2014.

[d]Both real-time (restricted use since September 2014, fully since July 2018) and intermittently scanned (reimbursed since July 2016) continuous glucose monitoring is only for people living with type 1 diabetes.

variables, with exchangeable correlation structure (diabetes centre) and study group as a dichotomous explanatory variable.

The statistical significance of the trend over the years for prevalence of diabetic retinopathy and albuminuria 15 years post-diagnosis was tested using GEE as described above, with study period (defined as the midpoint of the audit year) as a continuous explanatory variable, adjusted for age (continuous) and sex (dichotomous). The GEE model predictions are presented with the corresponding 95% confidence interval (CI). The analysis was additionally conducted with further adjustments for haemoglobin A1C (HbA1c), LDL-cholesterol, systolic blood pressure (SBP), body mass index (BMI) and their combined effect.

For the pairwise comparison of prevalence of diabetic retinopathy and albuminuria, the GEE model and analysis described above were repeated with study group and/or age at diabetes onset as categorical explanatory variables. Comparisons were adjusted using the Tukey method.

All *p*-values were two-sided; *p*-values <0.05 were considered statistically significant. Data analyses were performed using SAS software version 9.4 (SAS Institute, USA).

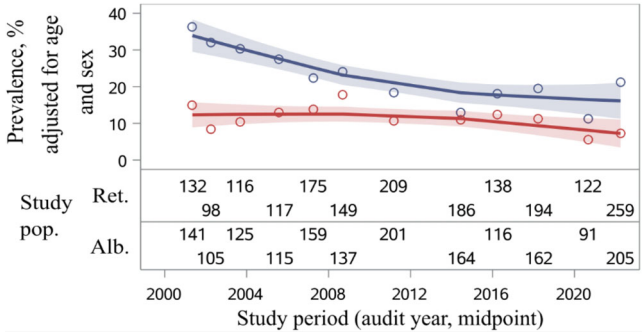

FIGURE 1 The evolution of the prevalence of diabetic retinopathy (blue circles, $N = 1895$) and albuminuria (red circles, $N = 1721$) 15 years post-diagnosis over the years, expressed as midpoint of the Initiative for Quality Improvement and Epidemiology in Diabetes audit year, adjusted for age and sex. The solid lines are the fitted locally estimated scatterplot smoothing (LOESS) curves, the shaded bands represents the 95% confidence interval of the fitted LOESS curve. The size of the study population in each audit year is indicated below the graphs.

3 | RESULTS

3.1 | Population characteristics by study group

Table 1 presents the comparative population characteristics by study group. Individuals in group A were older than those in group B (32.7 ± 7.6 years in group A vs. 31.6 ± 7.8 years in group B, $p < 0.001$), had a shorter duration of diabetes (14.9 ± 1.2 years in group A vs. 15.2 ± 1.1 years in group B, $p < 0.0001$), and a higher age at diabetes onset (17.8 ± 7.6 years in group A vs. 16.4 ± 7.7 years in group B, $p < 0.0001$). Glycaemic control was poorer in group A (HbA1c: 8.3 ± 1.6% [67.4 ± 17.9 mmol/mol] in group A vs. 8.1 ± 1.5% mmol/mol [64.6 ± 15.9 mmol/mol] in group B, $p < 0.0001$) as was the lipid profile (LDL-cholesterol: 2.6 ± 0.8 mmol/L in group A vs. 2.5 ± 0.8 mmol/L in group B, $p < 0.001$), whereas obesity prevalence was higher in group B (12.8% in group A vs. 17.4% in group B, $p < 0.01$). Group A had a higher rate of antihypertensive therapy use (14.9% in group A vs. 11.5% in group B, $p < 0.01$), while lipid-lowering therapy was more common in group B (9.2% in group A vs. 15.9% in group B, $p < 0.0001$). CGM was only available in group B, which also had a higher prevalence of CSII use (1.0% in group A vs. 13.8% in group B, $p < 0.0001$). Similar trends in population characteristics between the two study groups were observed in the different age at diabetes onset categories (Table S1).

3.2 | Evolution of the prevalence of diabetic retinopathy and albuminuria over time

Absence or presence of diabetic retinopathy and albuminuria could be defined for 1895 and 1721 individuals, respectively. In the total study population, the prevalence of diabetic retinopathy 15 years post-diagnosis, adjusted for age and sex, decreased from 36.3% (95% CI 27.9–44.7) in 2001 to 21.1% (95% CI 16.1–26.1) in 2022 ($p < 0.0001$) (Figure 1). This decrease remained significant upon additional adjustment for HbA1c, LDL-cholesterol, SBP, BMI and their combined effect (Table S2).

The prevalence of albuminuria, adjusted for age and sex, decreased from 14.9% (95% CI 9.0–20.8) in 2001 to 7.3% (95% CI 3.8–10.9) in 2022 ($p < 0.05$). This decrease remains present after correction for LDL and SBP and disappeared after correction for HbA1c and BMI (Table S2).

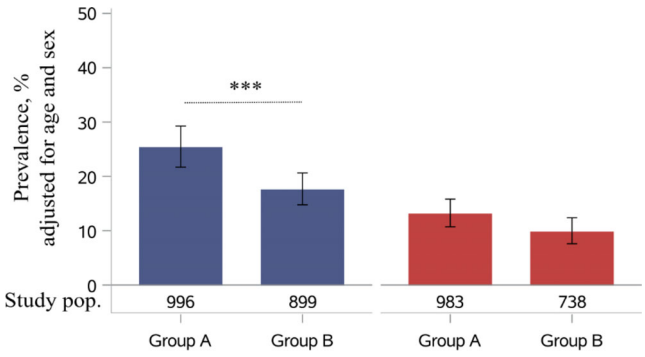

FIGURE 2 The prevalence of diabetic retinopathy (blue bars) and albuminuria (red bars) 15 years post-diagnosis, according to the study group, adjusted for age and sex. The size of the study groups are indicated at the bottom of the graph. ***$p < 0.001$ between study groups.

3.3 | The prevalence of diabetic retinopathy and albuminuria by study group

Figure 2 shows the prevalence of diabetic retinopathy and albuminuria 15 years post-diagnosis by study group, adjusted for age and sex. In group A, the prevalence of diabetic retinopathy was significantly higher compared to group B (25.3% [95% CI 21.7–29.3] vs. 17.5% [95% CI 14.8–20.6], $p < 0.001$), as was the prevalence of albuminuria although no longer statistically significant (13.0% [95% CI 10.7–15.8] vs. 9.7% [95% CI 7.6–12.4]). The prevalence of diabetic retinopathy remained significantly lower in group B compared to group A upon additional adjustment for either HbA1c, LDL-cholesterol, SBP, BMI or their combined effect (Table S3).

3.4 | Effect of age at diabetes onset on the prevalence of diabetic retinopathy and albuminuria by study group

In group A, the prevalence of diabetic retinopathy 15 years post-diagnosis adjusted for sex significantly increased by increasing age at diabetes onset: from 15.5% (95% CI 11.6–20.4) in those aged 0–11 years at diabetes onset to 26.8% (95% CI 20.8–33.7) in those aged 12–17 years at diabetes onset ($p < 0.05$) and further to 32.1% (95% CI 27.4–37.3) in those aged 18–29 years at diabetes onset ($p < 0.001$ vs. 0–11 years at diabetes onset category) (Figure 3). This increase remained present after correction for HbA1c, SBP and BMI, but after correction for LDL remained only significant between those aged 0–11 and 18–29 years at diabetes onset (Table S4). In group B, the prevalence of diabetic retinopathy remained comparable over the three age at diabetes onset categories (14.4% [95% CI 10.5–19.3], 17.0% [95% CI 12.4–22.8], 20.9% [95% CI 16.6–26.0] respectively, not significant).

Between study groups, the prevalence of diabetic retinopathy in the oldest age at diabetes onset category significantly decreased from 32.1% (95% CI 27.4–37.3) in group A to 20.9% (95% CI 16.6–26.0) in group B; $p < 0.05$. This decrease remained present after correction for SBP and BMI but disappeared after correction for HbA1c and LDL (Table S4).

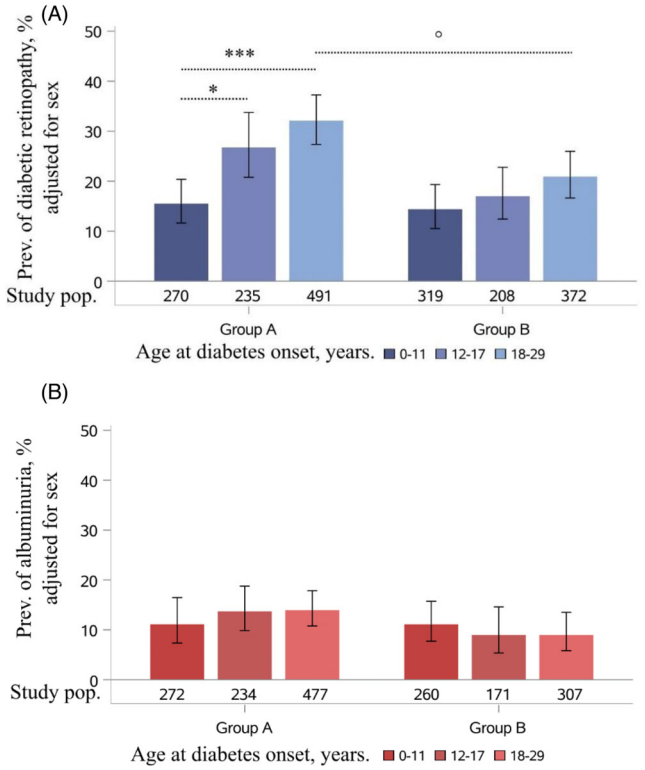

FIGURE 3 The prevalence of diabetic retinopathy (panel A, blue bars) and albuminuria (panel B, red bars) 15 years post-diagnosis, according to the study group and age at diabetes onset. The size of the study populations are indicated at the bottom of the graph. *$p < 0.05$, ***$p < 0.001$ between age at diabetes onset categories, per study group;°$p < 0.05$ between study groups, per age at diabetes onset category.

In both study groups, the prevalence of albuminuria 15 years after diabetes onset, adjusted for sex, was not impacted by the age at diabetes onset (Figure 3).

4 | DISCUSSION

We studied a real-world population of people living with T1D and demonstrated an impact of the era of diabetes onset on the prevalence of microvascular complications 15 years post-diagnosis, showing a decline in prevalence of diabetic retinopathy and albuminuria among those with diabetes onset after 1998.

In our study population, the prevalence of diabetic retinopathy and albuminuria 15 years post-diagnosis significantly declined over the years, and—especially for diabetic retinopathy—was lower in cohorts with later age at diabetes onset. In general, approximately one-third of the adult population living with diabetes develops retinopathy.[1] The prevalence of diabetic retinopathy in our study population is in line with the literature.[12–16] Approximately one-fourth of individuals with diabetes onset between 1985 and 1998 had developed diabetic retinopathy 15 years after diabetes onset, whereas this proportion declined to about one-fifth among those with diabetes onset between 1998 and 2009. The most significant reduction in diabetic retinopathy prevalence was observed among individuals with diabetes onset after 11 years of age.

Our findings align with previous reports showing a decline in diabetic retinopathy prevalence over time, likely due to advancements in diabetes management, increased awareness and the implementation of structured diabetes care programmes.[1,12,17–21] In Belgium, diabetes care is nationally structured through a multidisciplinary specialist care programme for individuals receiving intensive insulin therapy, known as the Diabetes Convention.[8] This system emphasised the early adoption of effective glucose management strategies, including CSII and CGM, while also promoting overall metabolic control through smoking reduction, improving blood pressure and lipid management, as well as patient education. In a previous study, we have shown that since 2012 glycaemic control in the Belgian population with T1D has improved at all ages supported by higher adoption of CSII and CGM.[8,22] Also in this study, the proportion of individuals with a HbA1c value <7.5% was lower in group A compared to group B (31.0% vs. 39.2%, $p < 0.001$), whereas the proportion of people with a HbA1c value ≥9% was higher (28.3% vs. 23%, $p < 0.01$). In addition, our data indicated improvements in lipid control among those with diabetes onset after 1998. The proportion of people with an LDL-cholesterol value <70 mmol/L was smaller in group A compared to group B (13.5% vs. 18.5%, p < 0.01), which can be partly attributed to increased use of more (effective) lipid-lowering therapy in response to the changing guidelines concerning lipid control in people living with diabetes like the European Association for the Study of Diabetes (EASD) and the American Diabetes Association (ADA) consensus guidelines recommending recently more routinely cardiovascular treatment in addition to glucose-lowering treatment in people living with diabetes.[8,22]

We saw a decline in the prevalence of albuminuria 15 years post-diagnosis over time. However, this difference was no longer significant when comparing our two study groups. In our analyses, the prevalence of albuminuria 15 years post-diagnosis decreased from 13.0% in study group A (diabetes onset between 1985 and 1998) to 9.7% in study group B (diabetes onset between 1998 and 2009) after correction for age and sex. This prevalence aligns with the prevalence reported in the Swedish National Diabetes Registry.[23,24]

The decline in albuminuria prevalence likely occurred in earlier decades, as supported by findings from previous studies. Hovind et al. reported a significant reduction in the cumulative incidence of diabetic nephropathy (defined as albuminuria (albumin excretion rate ≥30 mg/24 h), diabetes duration >10 years, presence of diabetic retinopathy, and absence of other kidney or renal tract disease) with more recent diabetes onset. The cumulative incidence of nephropathy at 20 years post-diagnosis decreased from 31.1% in individuals with diabetes onset between 1965 and 1969 to 13.7% in those with diabetes onset between 1979 and 1984 ($p = 0.004$).[18] Similarly, Nordwall et al. observed—with more recent diabetes onset—a decrease in the cumulative incidence of nephropathy (defined as albumin ≥300 mg/L) at 20 years post-diagnosis, from 30.3% in the cohort with diabetes onset between 1961 and 1965 to 9.2% for the cohort with diabetes onset between 1971 and 1975 ($p = 0.02$).[19] Also, Sigfrids et al. demonstrated a significant decrease in 25-year cumulative incidence for severe albuminuria (defined as albumin excretion rate ≥ 300 mg/24 h) from 26.8% for the 1970–1979 and 12.0% for 1980—diabetes onset cohorts ($p < 0.001$), with no further improvement in the 1990–1999 diabetes onset cohort.[25] In addition to improvements in glycaemic control, these previous studies attributed the significant decline in albuminuria to diagnostic tools and enhanced overall metabolic management, including adjustments in blood pressure targets, the availability of Renin-Angiotensin-Aldosterone System (RAAS) blockers and reductions in smoking behaviour.

The introduction of RAAS blockers occurred before our study period, which may explain the moderate reduction in albuminuria prevalence in our findings. In both this study and our previous research, we did not observe any changes in blood pressure control.[8] The use of antihypertensive therapy even declined slightly from 14.9% in group A to 11.5% in group B ($p < 0.01$), potentially indicating a shift towards more effective treatment strategies. However, the IQED database does not allow us to assess the brand or dosage of drugs used, neither the time between diabetes onset and the initiation of antihypertensive therapy and/or adherence to treatment guidelines. Additionally, we found no significant improvement in smoking habits between group A (22.4%) and group B (20.6%). Even more concerning is the increase in obesity prevalence observed between the two time periods, a trend that has previously been observed in people living with T1D in Belgium.[22,26] This evolution is most probably due to a more liberal attitude towards diet in people living with T1D, as well as the pursuit of tighter glucose control. However, as we recently showed, this evolution is associated with a more adverse metabolic profile (i.e., lipids and blood pressure), which may contribute to adverse cardiovascular outcomes.[22] The follow-up period of 15 years

in the present study does not allow us to make solid conclusions on macrovascular complications due to too low numbers of events.

Another explanation of the moderate decrease in prevalence of albuminuria in our study might be the slightly lower HbA1c values in our population at baseline (8.3 ± 1.6% group A; 8.1 ± 1.5% group B) compared to the values reported by Hovind et al. (mean HbA1c value during 20 years of follow-up: 8.9 ± 0.1% in individuals with diabetes onset between 1965 and 1969; 8.5 ± 0.1% in those with diabetes onset between 1979 and 1984), suggesting that good metabolic control postpones the onset of albuminuria.

The strengths of our study lie in its real-world setting, the extensive size of our database and the availability of data dating back to 2001. The IQED database is representative of the total T1D population in Belgium. Treatment and follow-up in the diabetes convention have always been free-of-charge for people living with T1D in Belgium, resulting in almost all people living with T1D adhering to this convention. Consequently, selective referral based on diabetic late complications or socioeconomic status is unlikely.[18] Nevertheless, there are some limitations. We investigated the prevalence of diabetic retinopathy but have in our data no information about the severity of diabetic retinopathy. However, within the diabetes convention system, yearly follow-up by the ophthalmologist is monitored in IQED as a quality indicator. Furthermore, due to the cross-sectional nature of the IQED database, we could not calculate incidence rates.

In conclusion, individuals diagnosed with T1D after 1998 had a lower prevalence of microvascular complications 15 years post-diagnosis compared to those diagnosed 10 years earlier, with the most pronounced decline observed in diabetic retinopathy among those with diabetes onset after the age of 11 years. The declining rates of diabetic retinopathy across different diagnostic eras underscore the effectiveness of measures in our health care system promoting the early introduction of novel technologies, like CGM, alongside efforts to enhance overall metabolic control through smoking reduction, optimisation of blood pressure and lipid management and emphasis on patient education.

AUTHOR CONTRIBUTIONS

AL, CM, CDB, PO and FN developed the concept and design of this study. Data analysis was performed by AL. AL, CM, CDB, PO, FN, J-CP, MV and LC made substantial contributions to the interpretation of results. AL, CM, CDB, PO and FN drafted the manuscript, and AL, CM, CDB, PO, FN, J-CP, MV and LC contributed to the critical revision of the manuscript for important intellectual content. AL, CM, CDB, PO, FN, J-CP, MV and LC approved the final manuscript for publication. AL had full access to the data and accepts the responsibility for the integrity of the data and the accuracy of the data analysis.

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CONFLICT OF INTEREST STATEMENT

All authors declare that no competing financial interests exist.

PEER REVIEW

The peer review history for this article is available at https://www.webofscience.com/api/gateway/wos/peer-review/10.1111/dom.70049.

DATA AVAILABILITY STATEMENT

Data cannot be shared publicly because of the use of pseudonymised person-level data. Actors wanting to access (parts of the) data are required to submit a request through the Belgian Health Data Agency (https://www.hda.belgium.be/en/data_request). An approval from the Belgian Information Security Committee might be required. For more information about the access procedure: iqed@sciensano.be. Metadata (e.g., overview of variables and legal framework) are available on https://fair.healthdata.be/.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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