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Impact of continuous glucose monitoring on hospitalizations and glucose control in people with type 2 diabetes: real-world analysis

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Abstract

Aim: The real-world benefits of continuous glucose monitoring (CGM) in the broad type 2 diabetes (T2D) population are not well studied. Our study evaluated the impact of CGM use on health care resource utilization over 12 months in adults with T2D.

Materials and Methods: This retrospective cohort analysis used Optum's de-identified Market Clarity data of >79 million people to evaluate CGM use in people with T2D who were treated with non-insulin (NIT), basal insulin (BIT) and prandial insulin therapy (PIT). The primary outcomes were changes in all-cause hospitalizations, acute diabetes-related hospitalizations and acute diabetes-related emergency room visits during the 6- and 12-month post-index period following transition from blood glucose monitoring to CGM. A pre-specified subgroup analysis assessed glucose control and medication changes among people with T2D over 1 year.

Results: The analysis included 74 679 adults with T2D (NIT; $n = 25\,269$), (BIT; $n = 16\,264$) and (PIT; $n = 33\,146$). Significant reductions in all-cause hospitalizations, acute diabetes-related hospitalizations and acute diabetes-related emergency room visits were observed in the 6-month post-index period that were sustained during the 6–12 month post-index period (NIT, −10.1%, −31.0%, −30.7%; BIT, −13.9%, −47.6%, −28.2%; and PIT, −22.6%, −52.7%, −36.6%, respectively). A subgroup analysis of 6030 people showed mean glycated haemoglobin reductions at approximately 3 months, which were also sustained throughout the post-index period: NIT, −1.1 (0.05)%; BIT, −1.1 (0.06)%; and PIT, −0.9 (0.04)%, $p < 0.0001$.

Conclusions: CGM use in real-life across different therapeutic regimens in adults with T2D was associated with reductions in health care resource utilization with improved glucose control over 1 year.

KEYWORDS
continuous glucose monitoring, continuous glucose monitoring, diabetes-related events, hospitalizations, insulin, multiple daily injections, non-insulin

1 | INTRODUCTION

Maintaining optimal glycaemia is essential in preventing and/or delaying the micro- and macrovascular complications of diabetes.[1–4] However, achieving the American Diabetes Association and European Association of Study for Diabetes recommended glycaemic control targets in the real world is difficult for many people with diabetes. More than 50% of adults with type 2 diabetes (T2D) have glycated haemoglobin (HbA1c) levels >8.0%.[5]

Recent data have also shown that the increasing prevalence of diabetes in the United States continues to drive a steady rise in health care resource utilization (HCRU).[6] The number of diabetes-related inpatient hospitalization days and emergency room visits rose from 29.8 million in 2017 to 42.1 million in 2022.[6] A similar increase in all-cause inpatient days among people with diabetes was also reported during the same period, from 40.3 million to 48.6 million.[6] Combined, these events comprise 62.3% of the direct medical expenditures of $421.9 billion attributable to diabetes in 2022.[6]

Despite recommendations, blood glucose monitoring use is suboptimal.[7,8] An increasing number of people with diabetes have now adopted CGM, in part because of improvements in accuracy, reliability and user convenience. Numerous studies have shown the clinical value of CGM in intensively treated people with multiple daily insulin injections or insulin pumps.[9–13] In addition to improvement in overall glycaemia, CGM use has also been shown to reduce hypoglycaemia, which is a major factor for delaying the treatment intensification for both people with type 1 diabetes (T1D) and people with T2D.

Although insurance coverage for CGM for people on any insulin treatment has been widely adopted, coverage for people treated with non-insulin medications in T2D is limited in the United States. In a recent narrative review of 29 randomized controlled trials and real-world studies, CGM use significantly improves HbA1c with reductions in hypoglycaemia in people with T2D treated with either intensive insulin regimens, non-intensive insulin (basal only) therapy, or non-insulin medications.[14] However, most of these studies included small sample sizes, <200 participants.

We evaluated the impact of CGM use on HCRU over 1 year in a large sample of adults with suboptimally controlled T2D who were treated with non-insulin therapy (NIT), basal insulin therapy (BIT) and prandial insulin therapy (PIT).

2 | MATERIALS AND METHODS

2.1 | Study data source

In this retrospective analysis, we utilized administrative claims from Optum's de-identified Market Clarity data (one of the largest commercially available databases in the United States that includes EMR and claims data), which included >79 million people in the United States electronic medical records (EMRs) from 2007 to 2023. Data from a subset of people were used along with their medical and claims history based on having at least one *International Classification of Diseases* (ICD10/ICD9) diagnosis code for diabetes. Data from a subset of people were also used along with their medical and claims history based on having clinical activity with ICD10 diagnosis codes E08.x through E13.x and O24.x (or the corresponding ICD9 equivalents). The compilation of claims and medical records was loaded onto an Amazon Redshift server and analysed using SAS 9.4. As the data contain both EMR and insurance payment data, we referred to a specific EMR record as an entry and an insurance payment as a claim.

The data consisted of all 5.6 million patients with diabetes with entries or claims between 1 January 2019 and 31 December 2022, whereas the study period was defined as any entry or claim between 30 June 2019 and 5 January 2022. In total, 74 679 adults with T2D met all the criteria (HbA1c between 7.0% and 15.0%, more than one CGM claim between 30 June 2019 and 5 January 2022) (Figure S1). The index date for this analysis was a person's first claim for a CGM device (sensor, transmitter, or receiver) in the study period. A person's pre-index period was defined as the 6 months before their index date and their corresponding post-period is the 12-month period following their index date, inclusive of their index date. Thus, the earliest someone could be included in the study was 30 June 2019, and the latest date that an individual could be included in the study was 5 January 2022.

The database contains underlying patient-level data for a defined population based on a set of selection criteria. The data included demographic information, medical records, laboratory data and pharmacy claims. The data for this study were extracted on 10 July 2023. ICD10 billing codes obtained from EMRs were used to identify people with T2D. Diabetes type was determined from the closest relevant diagnosis before the CGM claim, irrespective of the brand prescribed. National Drug Code (NDC) data was used to identify people who were treated with NIT, BIT and PIT with a CGM claim. To ensure that people were naïve to CGM, we excluded those with any evidence of a previous CGM claim (including sensor, transmitter, or receiver) identified via either NDC or Healthcare Common Procedure Coding System (HCPCS) codes in their pre-index period. Claims for the CGM were identified through either the presence of associated NDC codes or the appearance of the system name in the claim description field.

2.2 | Study population

In addition to having an index date during the study period, diagnosis of T2D was required. Other inclusion criteria were age ≥18 years on 31 December of the year before their index date and a claim for diabetes therapy in their pre-index period. There were no constraints imposed on any HbA1c values for those participants in the hospital and emergency room analyses. Diabetes type was determined from the closest relevant ICD10 diagnosis before the CGM claim for devices commercially available. If the diagnosis code was E10.x, they were classified as having T1D and they were excluded from the analysis. Similarly, if the closest previous relevant diagnosis code was E11.x, then they were classified as having T2D. If they had both E10.x and E11.x diagnosis codes on the same date in their pre-period, they were

excluded from the analysis. All study participants were required to be continuously enrolled in a health insurance plan throughout their pre- and post-index periods based on Optum's de-identified Market Clarity definition of continuous enrolment. The definition permits up to a 30-day gap in insurance coverage. Exclusion criteria included: a claim or evidence for any CGM (professional, personal, or implantable) during the pre-index period; a claim or evidence of using an insulin pump; or pregnancy during the pre- or post-index periods.

Individuals included in the glucose control analysis were required to have at least one HbA1c value in their pre-period and at least one HbA1c value in the 6-month to 12-month post-index period. Patients were stratified by pre-index period diabetes therapy: NIT, BIT and PIT with or without non-insulin medications. Individuals on a pre-mix insulin regimen in the pre-period were included in the baseline PIT group (Figure S1).

2.3 | Procedures

The study included two observation periods: a pre-index period, which included the 6 months before each person's first CGM claim; and a post-index period of 12 months subsequent to the first CGM claim. The index date was identified as the date of the first CGM claim between 30 June 2019 and 5 January 2022. Baseline HbA1c was defined as the value in the pre-index period closest to the index date (Figure S2).

2.4 | Outcomes

The primary outcomes of the study were changes in all-cause hospitalizations (ACH), acute diabetes-related hospitalizations (ADH) and acute diabetes-related events requiring emergency room (ADER) visits during the post-index period. ADH included hypoglycaemia, hypoglycaemic coma, clinical hyperglycaemia, diabetic ketoacidosis and hyperosmolarity. These were identified as either inpatient events with the associated ICD10 code or emergency outpatient events, which included emergency department services with the associated ICD10 code in any position. The ICD10 codes for acute events were as follows: hypoglycaemia (E16.1, E16.2, E10-11.649, E13.649), hypoglycaemic coma (E11-11.641, E13.641), hyperglycaemia (E10-11.65, E13.65), diabetic ketoacidosis (E11.1x, E13.1x) and hyperosmolarity (E11.00, E13.0x). For each patient, medical billing codes associated with the same service or admission date were counted as a single event.

Secondary outcomes included a pre-specified subgroup analysis to assess (a) change and sustainability of the glucose control (HbA1c) at 6 and 12 months, and (b) changes in medication classes during the post-index period. There were 6030 people with T2D where pre- and post-index period values were available in the dataset. The lack of values being recorded in the EMRs was the limitation of the availability of HbA1c values. Thus, the subgroup was not a 'selected' subgroup based on some characteristics but was likely because of missing values at random.

2.5 | Statistical analysis

All events (ACH, ADH, ADER) were grouped into three time periods: 6 months before index date; 179 days post-index date (inclusive of index date) and 6–12 months post-index date (annotated 'h1', 'H1' and 'H2' respectively). To facilitate the hospital event analysis, hospital discharge and admit dates on the same day were considered a continuation of the same event. For the HbA1c change analysis, we similarly construct time periods in a patient's post-period in 90-day intervals and annotate them by the quarter after the index date: '+Q1', '+Q2', '+Q3' and '+Q4'. For each person, we recorded their average HbA1c in each quarter. If a person did not have an HbA1c lab result in a particular time period, their HbA1c value was recorded as missing. Based on the inclusion criteria stated above, each person was required to have an HbA1c value in either '+Q3' or '+Q4'. We report a person's baseline HbA1c as the person's HbA1c closest to their index date in their pre-period.

ACH, ADH and ADER were compared between 'h1' and 'H1' as well as event rates between 'h1' and 'H2' using non-linear mixed effects models with repeated measures to account for the possibility of multiple events per individual. Based on fit metrics, we assumed a Poisson distribution for the number of events and used repeated measures with unstructured covariance to account for potential correlation between a patient's events during the three time periods. Similarly, for the longitudinal analysis of HbA1c, we used linear mixed-effects models with repeated measures to examine changes in the HbA1c post-index date.

3 | RESULTS

In total, 74 679 people with T2D were included in the assessment for ACH, ADH and ADER. The baseline characteristics are shown in Table 1. In total, 6030 people with HbA1c values in the pre-index period and during the post-index period were included in the analyses of change and sustainability of glycaemic improvement and medication changes.

3.1 | Hospitalizations and acute diabetes-related events

Among the 74 679 people with T2D included in these analyses (during the 6-month pre-index period), there was a total of 14 147 ACH, which was significantly reduced at 6 and 12 months (−23.1% and −18.8%, respectively). Similarly, reductions in the numbers of ADH (−52.5% and −49.5%, respectively) and ADER (−35.5% and −34.4%, respectively), were observed during the post-index period.

Reductions in ACH were observed in all treatment groups during the first 6 months and at 12 months of the post-index period: NIT, −14.2% and −10.1%; BIT, −18.4% and −13.9%; and PIT, −26.9% and −22.6%, respectively (Figure 1A).

Larger reductions were observed in ADH: NIT, −33.6% and −31.0%; BIT, −47.4% and −47.6%; and PIT, −56.5% and −52.7%,

TABLE 1 Baseline patient characteristics ($n = 74\ 679$).

Characteristics	NIT ($n = 25\ 269$)	BIT ($n = 16\ 264$)	PIT ($n = 33\ 146$)
Index age, year (SD)	56.9 (11.91)	57.7 (11.92)	58.3 (12.62)
Female sex, *n* (%)	12 351 (48.9)	7931 (48.8)	16 990 (51.3)
Race, *n* (%)			
Asian	908 (3.6)	351 (2.2)	643 (1.9)
Black	3542 (14.0)	2487 (15.3)	5066 (15.3)
White	14 615 (57.8)	9716 (59.7)	19 961 (60.2)
Unknown	6204 (24.6)	3710 (22.8)	7476 (22.6)
Ethnic group, *n* (%)			
Hispanic	1667 (6.6)	1074 (6.6)	2211 (6.7)
Non-Hispanic	16 072 (63.6)	10 692 (65.7)	22 208 (67.0)
Unknown	7530 (29.8)	4498 (27.7)	8727 (26.3)
Geographic regions, *n* (%)			
Midwest	8747 (34.6)	6018 (37.0)	13 458 (40.6)
Northeast	6594 (26.1)	3561 (21.9)	7329 (22.1)
South	6472 (25.6)	4367 (26.9)	7861 (23.7)
West	2235 (8.8)	1583 (9.7)	2914 (8.8)
Other/unknown	1221 (4.8)	735 (4.5)	1584 (4.8)

Note: Numbers in parenthesis are percentages for non-insulin treated (NIT), Basal insulin treated (BIT) and prandial Insulin treated (PIT).

respectively (Figure 1B). When examined as a percentage of the reduction in ACH, reductions in ADH accounted for 35.8% and 46.5% of ACH reductions in the NIT group at 6 and 12 months post-index, respectively; 57.2% and 76.0% of ACH reductions in the BIT group at 6 and 12 months post-index, respectively; and 59.0% and 65.5% of ACH reductions in the PIT group at 6 and 12 months post-index, respectively.

Reductions in ADER were also observed (NIT, −30.1% and −30.7%; BIT, −33.4% and −28.2%; and PIT, −36.9% and −36.6%, respectively) (Figure 1C). Events per 100 person years for all patients at 6 months pre-index, 6 months post-index and 6–12 months post-index are as follows: ACH 469, 351, 381; ADE 184, 78, 84; and ADER 719, 451, 456, respectively (Table S2). Odds ratios and *p*-values for hospitalizations and events between time periods are presented in Table S1. All comparisons with respect to the pre-index period were significant at $p < 0.05$.

Eighty-seven per cent of ADH requiring hospitalizations or emergency room visits were for hyperglycaemia or diabetic ketoacidosis in the post-index period. Additional data related to the total number of patients and the distribution of hypoglycaemia and hyperglycaemia events can be found in Table S3.

3.2 | Secondary analyses

The mean (SEM) HbA1c values decreased by 0.9% at about 3 months and a sustained effect was seen up to 12 months (Figure 2). At baseline, the mean HbA1c values for the NIT, BIT and PIT treatment groups were 8.6 (0.04)%, 9.0 (0.05)% and 8.9 (0.03)%, respectively. Mean HbA1c values dropped significantly at approximately 3 months in all groups ($p < 0.0001$). These reductions were sustained throughout the post-index period at 12 months: NIT, −1.1 (0.05)%; BIT, −1.1 (0.06)%; and PIT, −0.9 (0.04)%, respectively, all ($p < 0.0001$). The demographics of the subgroup cohort are presented in Table S4.

Clinically significant reductions in HbA1c levels were observed in all treatment groups regardless of medication changes (Table 2). The greatest reductions in HbA1c levels were observed in NIT- and BIT-treated groups. These reductions were associated with a reduction in the number of medications prescribed. Within the PIT treatment group, the greatest reduction in HbA1c was observed in people with net zero therapy changes.

Across all therapy groups, we observed reductions in the percentage of patients treated with metformin, sulphonylurea and dipeptidyl peptidase 4 inhibitor medications during the post-index period (Table S5). Changes in use of thiazolidinedione medications were relatively flat. We saw an increase in glucagon-like peptide-1 receptor agonists and sodium–glucose cotransporter-2 inhibitor use across all treatment groups. We also observed insulin intensification and deintensification across all treatment groups.

In addition, our study period coincided with the COVID-19 pandemic, which could have contributed to higher rates of hospitalizations during the study period. We examined ACH, where a COVID-19 diagnosis was recorded. ACH with a COVID-19 diagnosis trended from 5.8% (overall) in the pre-index period to 6.3% (overall) in the post-index period (Table S6).

4 | DISCUSSION

In this real-world study, we report that the use of CGM is associated with a reduction in ACH, ADH and ADER in people with T2D regardless of their therapeutic regimen. As recently reported, the per capita cost associated with inpatient days among people with diabetes is

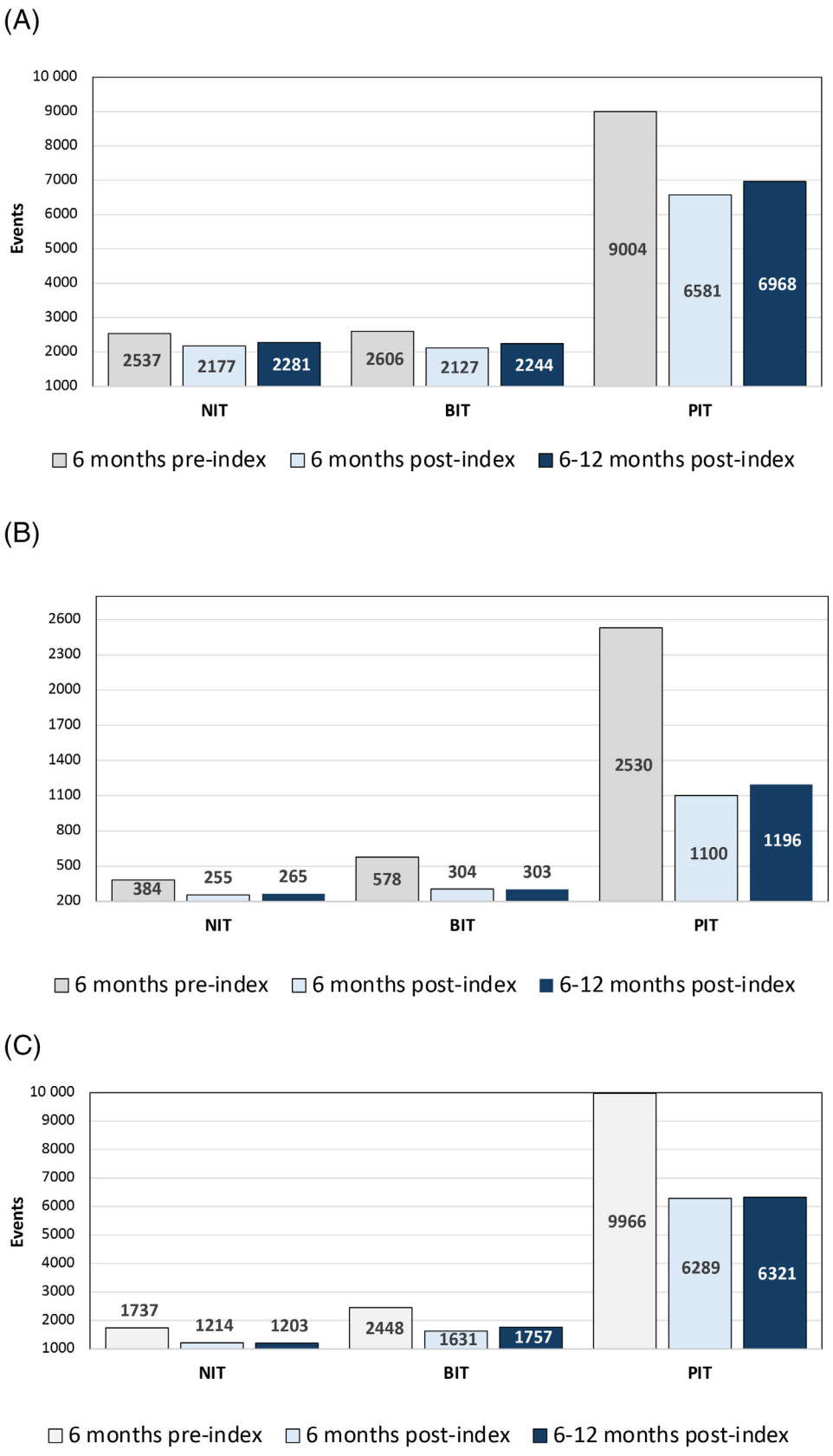

FIGURE 1 Changes in all-cause hospitalizations (ACH), acute diabetes-related hospitalizations (ADH), and acute diabetes-related emergency room visits (ADER) at 6- and 12-months post-index. The grey bars indicate the ACH, ADH and ADER events in the pre-index period. The light blue and dark blue bars show a significant reduction in events at 6 and 12 months, respectively, in the post-index period. BIT, basal insulin treatment; NIT, non-insulin therapy; PIT, prandial insulin therapy.

five-fold higher than for individuals without diabetes ($5668 vs. $1138, respectively).[6] In our study, CGM use was associated with reductions in ACHs during the first 6 months and was sustained up to 12 months. Larger reductions were observed in ADH and ADER, and reductions in ADH accounted for most (57.2–76%) of the ACH reductions among insulin users and 35.8%–46.5% of ACH among the NIT

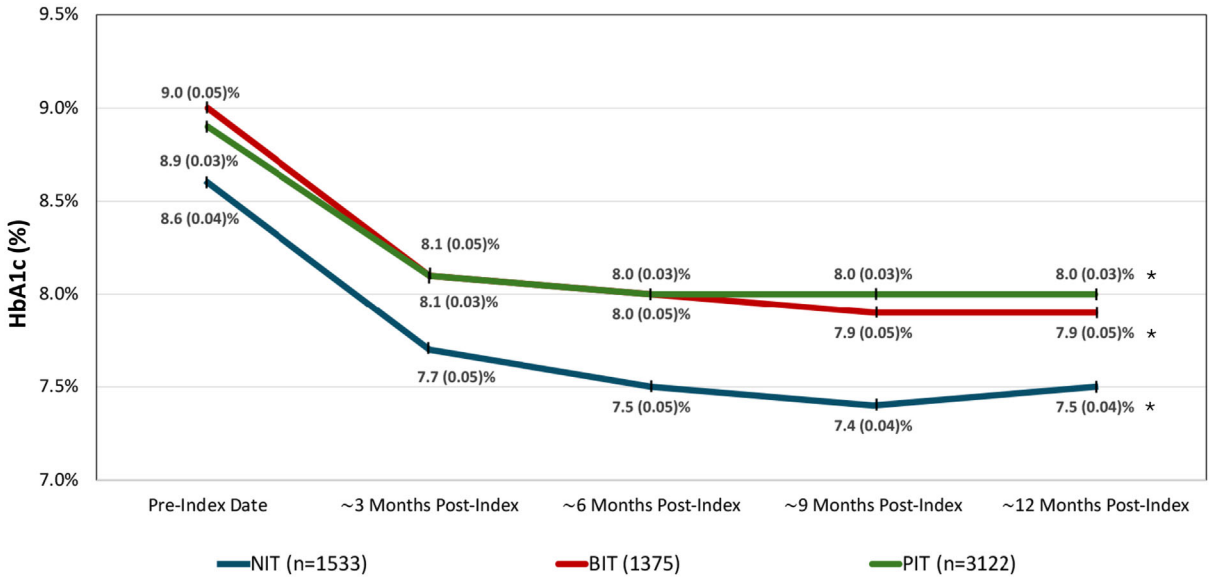

FIGURE 2 Improved glucose control associated with continuous glucose monitoring use at 3, 6 and 12 months. Non-insulin therapy (NIT), basal insulin treatment (BIT) and prandial insulin therapy (PIT) are represented in blue, red and green lines, respectively. A significant improvement was observed in glycated haemoglobin (HbA1c) as early as 3 months and was sustained throughout the study period. HbA1c values are represented as mean ± SEM. *$p < 0.0001$.

group. As expected, the reductions in ACH, ADH and ADER were significantly higher in the PIT group. Thus, CGM use may have helped to reduce acute diabetes complications, and the reduction in ACHs could be largely because of the reduction in diabetes-related hospitalizations.

A previous small study in people with T2D on rapid-acting insulin showed a decrease in ACH and ADH with the acquisition of CGM.[15] Another study that included both people with T1D and T2D showed a significant reduction in ADH, mainly from diabetic ketoacidosis.[16] Karter et al. reported a small decrease (−2.7%) in hypoglycaemia-related emergency room visits with no difference in hyperglycaemia-related events.[17] Our study included a larger sample size of adults with T2D on different therapeutic regimens.

We also reported a significant association between the claim for CGM and reductions in HbA1c values in all treatment groups. The HbA1c reduction occurred during the first 3 months and persisted throughout the study period (Figure 2). It was interesting that people treated with NIT at baseline achieved slightly greater reductions in HbA1c compared with BIT despite their lower baseline HbA1c levels. It is important to note that baseline HbA1c values were suboptimal in all three therapeutic groups (NIT, BIT and PIT). This finding supports the value and utility of CGM in non-insulin-treated people with T2D. This has also been shown in small, randomized trials and real-world retrospective and prospective studies.[18–22]

The strength of our study is the use of a large database to assess HCRU and changes in HbA1c levels across different therapeutic regimens over 1 year. Another strength is the additional subgroup analysis, which assessed glucose control and medication changes in approximately 6000 people during the post-index period. The number of patients included in this analysis was much smaller, as we pre-specified that HbA1c data had to be available in the pre-index period (6 months) and post-index period up to 1 year. However, if HbA1c was done as a point of care in the provider's office, the data were not available. Some of the changes observed in ACH, ADH and ADER could be attributed to the introduction of glucagon-like peptide-1 receptor agonists and sodium–glucose cotransporter-2 inhibitors. This will be the topic for future analysis. The significant HbA1c reduction without any medication changes across all therapy groups was noteworthy and possibly because of CGM use. Our findings would probably be generalizable to people with T2D with suboptimal glucose control treated with NIT and insulin therapies.

Our study has limitations. The use of an observational study design did not allow us to assess the significance of changes in HbA1c levels compared with similar cohorts with no CGM use. The ideal study design should have included a control group or a proper randomized control trial. As we had limited access to Optum's de-identified Market Clarity data, we chose to perform longitudinal analysis of ACH, ADH and ADER over a 1-year period after the CGM prescription, with the control period being a 6-month pre-index period. In addition, in the glucose control analysis only those subjects were included in the study where there was a pre- and post-index period HbA1c value available in the database. The lack of HbA1c values being recorded in the EMRs was the limitation.

The insurance claims did not provide the specific CGM prescribed or patient adherence, nor did we have access to CGM downloads for analysis. In addition, the data set did not provide potentially relevant demographic characteristics (e.g. socio-economic status, education level). We did have approximately 20% of the cohort represented by ethnic minorities. Clinical information regarding duration of diabetes, health care providers (e.g. specialists vs. primary care) and HbA1c

TABLE 2 Therapy adjustments by diabetes medication class.

	n (%)	Pre-index HbA1c	Post-index HbA1c	Post- vs. Pre-index HbA1c (SEM)	95% CI	*p*-Value
NIT ($n = 1533$)						
No medication changes	491 (32.0)	8.2	7.2	−0.9 (0.08)	−1.1035, −0.7914	<0.0001
Net zero medication changes[a]	154 (10.0)	8.6	7.6	−0.9 (0.15)	−1.2041, −0.6205	<0.0001
Medication classes added[b]	528 (34.4)	8.9	7.7	−1.2 (0.09)	−1.3804, −1.0361	<0.0001
Reduction of medications[c]	360 (23.5)	8.6	7.2	−1.4 (0.11)	−1.5754, −1.1482	<0.0001
BIT ($n = 1375$)						
No medication changes	377 (27.4)	8.6	7.7	−0.9 (0.08)	−1.0864, −0.7549	<0.0001
Net zero medication changes[a]	158 (11.5)	9.0	7.9	−1.1 (0.16)	−1.4282, −0.7957	<0.0001
Additional medication changes[b]	469 (34.1)	9.1	8.1	−1.0 (0.09)	−1.1887, −0.8399	<0.0001
Reduction of medications[c]	371 (27.0)	9.1	7.8	−1.3 (0.12)	−1.5134, −1.0420	<0.0001
PIT ($n = 3122$)						
No medication changes	1284 (41.1)	8.6	7.9	−0.7 (0.05)	−0.8085, −0.6254	<0.0001
Net zero medication changes[a]	261 (8.4)	9.4	7.8	−1.6 (0.15)	−1.8850, −1.3085	<0.0001
Additional medication changes[b]	481 (15.4)	9.2	8.1	−1.0 (0.09)	−1.2103, −0.8455	<0.0001
Reduction of medications[c]	1096 (35.1)	9.1	8.0	−1.2 (0.06)	−1.3008, −1.0488	<0.0001

Abbreviations: BIT, basal insulin treatment; CI, confidence interval; HbA1c, glycated haemoglobin; NIT, non-insulin therapy; PIT, prandial insulin therapy.
[a]Net zero medication class changes indicates equal number of medication classes were added and discontinued, not including the 'No medication changes'.
[b]Incremental medication class changes indicates ≥1 medication class added.
[c]Reduction of medication class change indicates ≥1 medication class discontinued. All data are reported as mean ± SEM.

assays (central laboratory vs. point-of-care) were also not available. The COVID-19-related admissions in the pre-index period did not confound our analysis regarding overall ACH. In fact, based on the slight increase in the percentage of ACHs with a COVID-19 diagnosis in the post-period, the association between CGM initiation and ACH may be understated (Table S5).

Despite these limitations, our findings support expansion of coverage of CGM use for people with T2D treated with NIT or less-intensive insulin therapies. This may help improve glycaemic control and reduce hospitalizations and overall health care costs. In summary, our study supports wider coverage for people with T2D who are generally not considered to be eligible.

In this real-world, retrospective study involving people with T2D, we observed a reduction in ACH, ADH and ADER. A sustained improvement in levels with CGM use was observed in suboptimally controlled glycaemia in adults with T2D treated with NIT, BIT and PIT.

AUTHOR CONTRIBUTIONS

SKG, IBH, RMB and ER conceptualized the study, interpreted the data, and wrote and edited the manuscript. CP and JSB analysed and interpreted the data and edited the manuscript. BU interpreted the data and edited the manuscript. All authors contributed to the discussion, reviewed the manuscript, and approved the final version of the manuscript. SKG is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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CONFLICT OF INTEREST STATEMENT

SKG has received consulting fees and honoraria for participation on advisory boards for Medtronic, Roche Diabetes Care, Merck, Lexicon, Novo Nordisk, Sanofi, MannKind, Senseonics, Zealand, and Eli Lilly and Company and research grants from Eli Lilly and Company, Novo Nordisk, Merck, Lexicon, Medtronic, Dario, the National Cancer Institute, T1D Exchange, the National Institute of Diabetes and Digestive and Kidney Diseases, JDRF, Animas, Dexcom, and Sanofi. IBH serves as an advisory board member for Abbott Diabetes Care, Roche, and Bigfoot, and GWave and receives research grant support from Dexcom. RMB has received research support, consulted, or has been on a scientific advisory board for Abbott Diabetes Care, Ascensia, CeQur Corporation, DexCom, Hygieia, Insulet, Johnson & Johnson, Lilly, Medtronic, Novo Nordisk, Onduo, Roche, Sanofi, United Healthcare and Zealand. ER, BU, CP are employed by Roche Diagnostics. JSB has no conflicts of interest to declare.

PEER REVIEW

The peer review history for this article is available at https://www.webofscience.com/api/gateway/wos/peer-review/10.1111/dom.15866.

DATA AVAILABILITY STATEMENT

The data are commercially available from Optum for a fee.

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REFERENCES

1. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose control with sulphonlylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). *Lancet*. 1998;352(9131):837-853.
2. Holman RR, Paul SK, Bethel A, Matthews DR, Neil HAW. 10-year follow-up of intensive glucose control in type 2 diabetes. *N Engl J Med*. 2008;359:1577-1589.
3. Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications of insulin-dependent diabetes mellitus. *N Engl J Med*. 1993;329:977-986.
4. Nathan DM, Cleary PA, Backlund JY, et al. Diabetes control and complications trial/Epidemiology of Diabetes interventions and complications (DCCT/EDIC) study research group: intensive diabetes treatment and cardiovascular disease in patients with type 1 diabetes. *N Engl J Med*. 2005;353:2643-2653.
5. Bin Rakhis SA Sr, AlDuwayhis NM, Aleid N, AlBarrak AN, Aloraini AA. Glycemic control for type 2 diabetes mellitus patients: a systematic review. *Cureus*. 2022;14(6):e26180. doi:10.7759/cureus.26180
6. Parker ED, Lin J, Mahoney T, et al. Economic costs of diabetes in the U.S. in 2022. *Diabetes Care*. 2024;47(1):26-43. doi:10.2337/dci23-0085
7. Puckrein GA, Hirsch IB, Parkin CG, et al. Assessment of glucose monitoring adherence in medicare beneficiaries with insulin-treated diabetes. *Diabetes Technol Ther*. 2023;25(1):31-38. doi:10.1089/dia.2022.0377
8. Siddiqui MH, Khan IA, Moyeen F, Chaudhary KA. Identifying barriers to therapeutic adherence in type 2-diabetes: a complex and multidimensional clinical issue. *Asp Biomed Clin Case Rep*. 2019;2(1):22-28.
9. Beck RW, Riddlesworth T, Ruedy K, et al. Effect of continuous glucose monitoring on glycemic control in adults with type 1 diabetes using insulin injections: the DIAMOND randomized clinical trial. *JAMA*. 2017;317:371-378.
10. Beck RW, Riddlesworth TD, Ruedy K, et al. Continuous glucose monitoring versus usual care in patients with type 2 diabetes receiving multiple daily insulin injections: a randomized trial. *Ann Intern Med*. 2017;167:365-374.
11. Šoupal J, Petruželková L, Grunberger G, et al. Glycemic outcomes in adults with t1d are impacted more by continuous glucose monitoring than by insulin delivery method: 3 years of follow-up from The COMISAIR study. *Diabetes Care*. 2020;43(1):37-43.
12. Haak T, Hanaire H, Ajjan R, et al. Use of flash glucose sensing technology for 12 months as a replacement for blood glucose monitoring in insulin-treated type 2 diabetes. *Diabetes Ther*. 2017;8:573-586.
13. Heinemann L, Guido Freckmann G, Gabriele Faber-Heinemann G, et al. Benefits of continuous glucose monitoring use in adults with type 1 diabetes and impaired hypoglycaemia awareness and/or severe hypoglycaemia treated with multiple daily insulin injections: results of the multicentre, randomised controlled HypoDE study. *The Lancet*. 2018;391(10128):1367-1377.
14. Aleppo G, Hirsch IB, Parkin CG, et al. Coverage for continuous glucose monitoring (CGM) for individuals with type 2 diabetes treated with non-intensive therapies: an evidence-based approach to policy-making. *Diabetes Technol Ther*. 2023;25(10):741-751. doi:10.1089/dia.2023.0268
15. Bergenstal RM, Kerr MSD, Roberts GR, Souto D, Nabutovsky Y, Hirsch IB. Flash CGM is associated with reduced diabetes events and hospitalizations in insulin-treated type 2 diabetes. *J. Endocr. Soc*. 2021;5(4):bvab013. doi:10.1210/jendso/bvab013 Accessed March 14, 2021.
16. Riveline JP, Roussel R, Vicaut E, et al. Reduced rate of acute diabetes events with flash glucose monitoring is sustained for 2 years after initiation: extended outcomes from the RELIEF study. *Diabetes Technol Ther*. 2022;24(9):611-618. doi:10.1089/dia.2022.0085
17. Karter AJ, Parker MM, Moffet HH, Gilliam LK, Dlott R. Association of real-time continuous glucose monitoring with glycemic control and acute metabolic events among patients with insulin-treated diabetes. *JAMA*. 2021;325(22):2273-2284.
18. Aronson R, Brown RE, Chu L, et al. Impact of flash glucose monitoring in people with type 2 diabetes inadequately controlled with non-insulin antihyperglycaemic therapy (IMMEDIATE): a randomized controlled trial. *Diabetes Obes Metab*. 2023;25(4):1024-1031. doi:10.1111/dom.14949
19. Wada E, Onoue T, Kobayashi T, et al. Flash glucose monitoring helps achieve better glycemic control than conventional self-monitoring of blood glucose in non-insulin-treated type 2 diabetes: a randomized controlled trial. *BMJ Open Diabetes Res Care*. 2020;8(1):e001115. doi:10.1136/bmjdrc-2019-001115
20. Welsh J, Grace T. Rapid HbA1c reductions in patients with type 2 diabetes in a community setting following initiation of real-time continuous glucose monitoring. In: *The official journal of ATTD advanced technologies & treatments for diabetes Conference 22-25 February 2023, Berlin & Online. Diabetes Technol Ther*. 2023;25(Suppl 2):EP149/#323.
21. Dowd R, Jepson LH, Green CR, Norman GJ, Thomas R, Leone K. Glycemic outcomes and feature set engagement among real-time continuous glucose monitoring users with type 1 or non-insulin-treated type 2 diabetes: retrospective analysis of real-world data. *JMIR Diabetes*. 2023;8:e43991. doi:10.2196/43991
22. Wright EE, Kerr MSD, Reyes IJ, Nabutovsky Y, Miller M. Use of flash continuous glucose monitoring is associated with A1C reduction in

people with type 2 diabetes treated with basal insulin or non-insulin therapy. *Diabetes Spectr*. 2021;34(2):184-189. doi:10.2337/ds20-0069

SUPPORTING INFORMATION

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

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