Safety and Glycemic Outcomes With a Tubeless Automated Insulin Delivery System in Very Young Children With Type 1 Diabetes: A Single-Arm Multicenter Clinical Trial

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OBJECTIVE

Very young children with type 1 diabetes often struggle to achieve glycemic targets, putting them at risk for long-term complications and creating an immense management burden for caregivers. We conducted the first evaluation of the Omnipod 5 Automated Insulin Delivery System in this population.

RESEARCH DESIGN AND METHODS

A total of 80 children aged 2.0–5.9 years used the investigational system in a single-arm study for 13 weeks following 14 days of baseline data collection with their usual therapy.

RESULTS

There were no episodes of severe hypoglycemia or diabetic ketoacidosis. By study end, HbA_{1c} decreased by 0.55% (6.0 mmol/mol) ($P < 0.0001$). Time with sensor glucose levels in target range 70–180 mg/dL increased by 10.9%, or 2.6 h/day ($P < 0.0001$), while time with levels <70 mg/dL declined by median 0.27% ($P = 0.0204$).

CONCLUSIONS

Use of the automated insulin delivery system was safe, and participants experienced improved glycemic measures and reduced hypoglycemia during the study phase compared with baseline.

Very young children with type 1 diabetes are completely reliant on others for management of their diabetes and are often unable to communicate their needs by self-identifying hypo- or hyperglycemia (1). Recent data highlight the struggle in achieving glycemic targets in this group (1–3). A diagnosis of type 1 diabetes at such a young age can have a profound and lasting impact, not only on the child's health (4,5), but also on the entire family (6).

Therapies with which practitioners aim to improve time in target range (TIR), such as automated insulin delivery (AID) systems, may alleviate some of these challenges, with several options available for those aged >6 years. Findings from

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**A list of members for the Omnipod 5 in Preschoolers Study Group can be found in Supplementary Material.*

studies of AID systems have demonstrated improvement in glycemia without increased self-care burden (7–9); however, exploration of this technology in very young children has been sparse (10,11). It is critical to study new therapies that may allow more targeted glycemia in this age-group.

The Omnipod 5 Automated Insulin Delivery System (Insulet Corporation) has previously been studied in those with type 1 diabetes aged 6–70 years (12). In this single-arm study, we assessed the safety and glycemic outcomes with this system in children aged 2.0–5.9 years with type 1 diabetes.

RESEARCH DESIGN AND METHODS

This single-arm, multicenter, prospective outpatient clinical study was conducted at 10 sites across the U.S. from August 2020 to January 2021. A 14-day standard therapy phase, wherein participants used their usual therapy for baseline continuous glucose monitoring (CGM) data collection, was followed by a 13-week AID study phase (see Supplementary Material for details).

Caregivers were trained on the use of the investigational device (Supplementary Fig. 1): a tubeless insulin pump (Pod) with embedded proprietary AID algorithm (Omnipod 5), interoperable CGM (Dexcom G6), and mobile application (Omnipod 5 app) on a locked-down Android phone (13). During the AID phase, the system delivered insulin microboluses every 5 min using a target glucose value (customizable between 110 and 150 mg/dL in 10 mg/dL increments by time of day). Follow-up visits were conducted every 2 weeks (in person = 5%, virtual = 95%) (Supplementary Table 1).

The protocol (clinical trial reg. no. NCT04476472, ClinicalTrials.gov) was approved by relevant local review boards and a central institutional review board. Oversight was provided by an independent data and safety monitoring board. Eligible participants were 2.0–5.9 years of age and diagnosed with type 1 diabetes, with HbA_{1c} <10% (86 mmol/mol) at screening. There was no minimum requirement for body weight or total daily dose (TDD) of insulin and no requirement of previous pump or CGM use. Key exclusion criteria were history of diabetic keto-acidosis (DKA) (unrelated to intercurrent illness, infusion set failure, or initial diagnosis) or severe hypoglycemia (SH) in the past 6 months (full criteria: Supplementary Table 2). Each participant's caregiver provided informed consent.

The primary safety end points were incidence rates of SH and DKA. The primary glycemic end points were HbA_{1c} at the end of the AID phase compared with baseline and TIR (70–180 mg/dL) during the AID phase compared with the standard therapy phase. Secondary end points with prespecified hypotheses were percent time with glucose level <70 mg/dL and >180 mg/dL during AID compared with standard therapy.

Glycemic end points were tested with paired *t* tests or Wilcoxon signed rank tests (the latter used for comparisons with <10 participants or if Shapiro-Wilk tests of normality were significant [$P < 0.05$]). The primary glycemic end points were tested independently with a two-sided 2.5% significance level. If at least one was significant, the secondary end points with prespecified hypotheses would be tested, with use of the Holm method to maintain a family-wise error rate at the two-sided 5.0% significance level. For additional end points a two-sided 5.0% significance level was used. Analyses were conducted with SAS, version 9.4.

RESULTS

A total of 80 participants were enrolled (Supplementary Table 3). All completed the study (Supplementary Fig. 2) and continued in the optional extension phase.

There were no episodes of SH or DKA during the AID phase. Prolonged hyperglycemia (blood glucose ≥300 mg/dL and ketones >1.0 mmol/L) occurred 20 times across 18.8% of participants (0.27 per 100 patient-days [Supplementary Table 4]). Of these events, 7 were deemed "possibly related" and 12 "related" to the study device, most likely due to infusion site issues; each resolved without progression to DKA.

Mean ± SD HbA_{1c} decreased from 7.4 ± 1.0% (57 ± 10.9 mmol/mol) at baseline to 6.9 ± 0.7% (52 ± 7.7 mmol/mol) at study end ($P < 0.0001$ [Supplementary Fig. 3]), and TIR increased from 57.2 ± 15.3% to 68.1 ± 9.0% ($P < 0.0001$), both meeting prespecified significance criteria (Table 1). Mean TIR was 61.3% and 67.8% for days 1–3 and 4–6 of AID. TIR increases were observed both overnight (0000–0600 h), from 58.2 to 81.0% ($P < 0.0001$), and during daytime (0600–2400 h), from 56.9 to 63.7% ($P < 0.0001$) (Supplementary Table 5). The percentage achieving HbA_{1c} <7.0% (53 mmol/mol) increased from 31 to 54%. The proportion achieving >70% TIR increased from 18 to 44%, while 83% achieved >60% TIR (Supplementary Table 6, Supplementary Fig. 4).

Time with glucose level >180 mg/dL decreased by mean ± SD 9.9 ± 10.5% ($P < 0.0001$) and time <70 mg/dL declined by a median of 0.27% (interquartile range −1.54, 0.46; $P = 0.0204$), both meeting the prespecified significance criteria. Additional outcomes are available in Supplementary Material (Supplementary Fig. 5, glucose profile; Supplementary Table 7, subgroup analyses; Supplementary Table 8, total daily dose, BMI).

Median time in automated mode during the 13-week AID phase was 97.8% (interquartile range 95.8, 98.5). The 110 mg/dL and 120 mg/dL target glucose settings were used most often, representing 33% and 42% of total study time, respectively (Supplementary Tables 9 and 10). There were ∼0.5 device deficiencies per person-month of system use.

CONCLUSIONS

This trial demonstrated the safety of the tubeless AID system in a group of very young children with type 1 diabetes. Participants also experienced improved glycemic outcomes and decreased time in level 1 hypoglycemia during the study phase compared with baseline. Children spent 2.6 more hours per day in target range. The proportion achieving the >70% TIR consensus goal increased by 2.5-fold, while more than four in five achieved the less stringent >60% TIR goal (14). Importantly, increased TIR did not come at the expense of additional hypoglycemia; rather, time with glucose level <70 mg/dL decreased by ∼4 min/day, and there were no episodes of SH or DKA, highlighting the safety of the system. HbA_{1c} decreased to 6.9% (52 mmol/mol), and the percentage achieving the American Diabetes Association–recommended target of HbA_{1c} <7.0% (53 mmol/mol) increased 1.7-fold (15).

Exploration of AID systems in very young children has been sparse. The single system available for use in this age-group in both the U.S. and Europe resulted in mean ± SD HbA_{1c} 7.5 ± 0.6%

Table 1—Primary and secondary glycemic outcomes (*N* = 80)

	Baseline or standard therapy phase‡	Follow-up or automated insulin delivery phase‡	Change	*P* value§
Overall (24 h)				
Primary glycemic end points:				
HbA_{1c}, %	7.4 ± 1.0, 7.4 (6.8, 8.1)	6.9 ± 0.7, 6.9 (6.5, 7.4)	−0.55 ± 0.58, −0.40 (−0.85, −0.10)	<0.0001
HbA_{1c}, mmol/mol	57 ± 10.9, 57 (51, 65)	52 ± 7.7, 52 (48, 57)	−6.0 ± 6.3, −4.4 (−9.3, −1.1)	<0.0001
% TIR 70–180 mg/dL	57.2 ± 15.3, 59.1 (48.0, 67.5)	68.1 ± 9.0, 68.4 (61.4, 74.1)	10.9 ± 9.6, 8.9 (4.9, 13.8)	<0.0001
Mean sensor glucose value, mg/dL	171.1 ± 30.5, 164.1 (148.6, 189.0)	157.4 ± 16.8, 155.4 (147.1, 170.6)	−13.7 ± 19.9, −9.5 (−17.5, −1.4)	<0.0001
SD of sensor glucose values, mg/dL	64.9 ± 13.4, 64.0 (56.0, 73.1)	59.6 ± 10.3, 59.5 (53.0, 66.2)	−5.3 ± 8.0, −4.6 (−9.3, −0.5)	<0.0001
Coefficient of variation of sensor glucose values, %†	38.1 ± 5.5, 37.4 (35.1, 41.7)	37.7 ± 4.0, 37.7 (35.1, 40.5)	−0.4 ± 4.2, −0.5 (−3.6, 2.3)	0.4232
% time in glucose range				
<54 mg/dL	0.81 ± 1.68, 0.24 (0.05, 0.84)	0.47 ± 0.54, 0.26 (0.16, 0.60)	−0.34 ± 1.33, 0.06 (−0.30, 0.16)	0.9394
<70 mg/dL	3.43 ± 3.87, 2.19 (0.89, 4.68)	2.46 ± 1.83, 1.94 (1.18, 3.43)	−0.97 ± 2.75, −0.27 (−1.54, 0.46)	0.0204
>180 mg/dL	39.4 ± 16.7, 37.0 (27.4, 50.0)	29.5 ± 9.8, 29.3 (23.1, 37.2)	−9.9 ± 10.5, −7.6 (−12.8, −3.5)	<0.0001
≥250 mg/dL	14.8 ± 12.1, 11.5 (5.4, 21.0)	9.2 ± 5.6, 8.4 (5.2, 13.0)	−5.6 ± 8.9, −2.3 (−6.6, −0.1)	<0.0001
≥300 mg/dL	6.0 ± 7.3, 3.5 (1.1, 8.3)	3.2 ± 2.8, 2.4 (1.2, 4.6)	−2.7 ± 6.1, −0.7 (−2.5, 0.2)	<0.0001
Overnight (0000–0600 h)				
Primary glycemic end point: % TIR 70–180 mg/dL	58.2 ± 18.7, 60.6 (47.8, 70.1)	81.0 ± 10.0, 82.4 (76.8, 88.7)	22.8 ± 14.8, 19.5 (12.8, 32.2)	<0.0001
Mean sensor glucose value, mg/dL	168.1 ± 33.3, 163.5 (147.6, 189.3)	140.7 ± 16.4, 141.1 (128.7, 150.3)	−27.4 ± 25.4, −22.9 (−44.5, −9.0)	<0.0001
SD of sensor glucose values, mg/dL	58.0 ± 14.0, 57.8 (50.1, 64.1)	45.5 ± 10.8, 45.7 (36.9, 52.0)	−12.5 ± 11.5, −11.0 (−20.5, −6.7)	<0.0001
Coefficient of variation of sensor glucose values, %†	34.7 ± 6.6, 35.2 (30.9, 38.8)	32.1 ± 5.2, 31.6 (29.2, 35.3)	−2.6 ± 6.7, −3.6 (−6.9, −0.3)	0.0002
% time in glucose range				
<54 mg/dL	0.85 ± 1.94, 0.00 (0.00, 0.97)	0.39 ± 0.53, 0.18 (0.06, 0.53)	−0.46 ± 1.78, 0.00 (−0.51, 0.13)	0.1128
<70 mg/dL	3.41 ± 4.79, 1.66 (0.40, 4.21)	2.13 ± 1.94, 1.58 (0.65, 2.89)	−1.28 ± 4.17, −0.44 (−2.17, 0.63)	0.0185
>180 mg/dL	38.4 ± 20.1, 36.5 (24.8, 51.1)	16.9 ± 10.3, 15.5 (8.4, 21.8)	−21.5 ± 16.0, −19.1 (−31.5, −11.3)	<0.0001
≥250 mg/dL	13.0 ± 13.2, 8.3 (3.4, 17.6)	3.9 ± 3.9, 3.1 (1.2, 5.0)	−9.1 ± 11.4, −5.1 (−13.8, −1.0)	<0.0001
≥300 mg/dL	4.3 ± 6.7, 1.3 (0.0, 5.6)	1.2 ± 1.6, 0.6 (0.1, 1.9)	−3.1 ± 6.1, −0.6 (−4.7, 0.0)	<0.0001

Data are means ± SD, median (interquartile range). To convert the values for glucose to millimoles per liter, multiply by 0.05551. TIR, time in target range. ‡Baseline and follow-up data were used for the primary glycemic end point of HbA_{1c}; the remaining outcomes are described for the standard therapy phase and the automated insulin delivery phase. §Unadjusted *P* value determined with two-sided paired *t* tests for overall coefficient of variation of sensor glucose, overnight SD of sensor glucose, and overnight % time with glucose level >180 mg/dL. †Coefficient of variation of sensor glucose is SD divided by the mean. Two-sided Wilcoxon signed rank tests were used for all other outcomes. Both primary glycemic end points (HbA_{1c} and % TIR) were considered significant at the prespecified 2.5% cutoff. Thus, the secondary outcomes with prespecified hypotheses were tested. The first secondary glycemic end point with prespecified hypothesis (% time with glucose level >180 mg/dL) was considered significant at the prespecified 2.5% cutoff and the second secondary end point with prespecified hypothesis (% time with glucose <70 mg/dL) was considered significant at the prespecified 5.0% cutoff. All other end points were considered significant at the 5.0% cutoff.

(58 ± 6.6 mmol/mol) and increase in TIR to 63.8% (increase of 8.1%), while mean time with glucose level <70 mg/dL was unchanged at 3.2% (10). In a recent evaluation of the other system available in this age-group (Europe only), TIR was 71.6 ± 5.9%, while median time <70 mg/dL was 4.9% (11).

Limitations of this study include the single-arm design, which could result in overestimation of improvement in glycemic outcomes as it does not account for potential improvements through study-related interactions (every 2 weeks). Despite broad eligibility criteria, some characteristics of the study population, such as exclusion of participants with recent severe glycemic events (implemented for safety reasons) and the relative homogeneity (77.5% White non-Hispanic, many with near-target glycemia as measured by HbA_{1c}, and the majority had prior technology experience), may make the findings less generalizable, and additional data are needed from more diverse groups of young children. A strength of this study is that prior pump experience was not required for inclusion.

With the goal of examining the use of a tubeless on-body AID system in very young children, the results of this trial indicated safety of the system observed alongside improvements in glycemia. AID systems may provide the opportunity to attain treatment goals, with benefits reaped beyond numbers, and the chance to reduce the risk of acute and chronic complications, especially given the long duration of diabetes these children will have.

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