Intensive management from diagnosis improves HbA1c at 12 months post-diagnosis: results from a prospective cohort study in children with newly diagnosed type 1 diabetes

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ABSTRACT

AIMS: To examine the impact of intensive management of type 1 diabetes (T1D) from diagnosis on HbA_{1c} 12 months from diagnosis.

METHODS: HbA_{1c} measured 12 months after diagnosis for 70 consecutively newly diagnosed children with T1D following implementation of an intensive management protocol was compared with 70 children consecutively diagnosed immediately pre-implementation. Intensive management involved carbohydrate counting and flexible insulin dosing from first meal with subcutaneous insulin, targeted blood glucose levels from 4–8mmol/L irrespective of time of day, avoidance of twice daily insulin regimen and promotion of continuous glucose monitoring (CGM). HbA_{1c}, diabetes technology use and insulin regimen at 12 months post-diagnosis were compared.

RESULTS: The post-intensive management implementation cohort had an improved mean HbA_{1c} of 58.2±15.3mmol/mol vs 63.7±10.7mmol/mol at 12 months (p=0.014). The proportion of young people with diabetes meeting a target HbA_{1c} of <53mmol/mol at 12 months improved from 11% to 40% (p=<0.001). There was a reduction of twice daily insulin regimen from 66% to 11% (p=<0.001), and increased CGM use from 57% to 76% (p=0.02).

CONCLUSION: Intensive management when implemented with consistent messaging from the multi-disciplinary team resulted in clinic-wide improvements in HbA_{1c} and the proportion meeting HbA_{1c} targets.

Type 1 diabetes (T1D) is a demanding, life-long journey for affected people and their caregivers. Management is aimed at reducing long-term complications while avoiding episodes of hypoglycaemia. The landmark Diabetes Control and Complications Trial (DCCT) has shown that an intensive insulin regimen with multidisciplinary team support is the most effective way of achieving this for both microvascular and macrovascular complications of T1D.[1] As set out in the International Society for Paediatric and Adolescent Diabetes (ISPAD) clinical practice guidelines from 2022, the target HbA_{1c} for young people with diabetes is <53 mmol/mol (<7.0%).[2] However, two recent Australasian studies have demonstrated widespread and persistent sub-optimal glycaemic control, with only 27% of children and 12.3% of adolescents achieving the recommended HbA_{1c} levels.[3,4]

There is evidence that it is important to establish glycaemic control early and that there is a window of opportunity at the time of diagnosis to optimise this control. This is because the trajectory of patient HbA_{1c} typically decreases over the first 5 to 6 months post-diagnosis, and then rises to a steady state around 12–18 months.[5,6] Subsequently, an individual's long-term HbA_{1c} trend rarely alters beyond 5 years post-diagnosis.[7] It is possible, therefore, that intensive management in the first 6 months following diagnosis could have a long-term impact on glycaemic outcomes.

Despite this finding, and the well-established efficacy of intensive management in T1D, there is still some variation in the approach from diagnosis. A survey of 100 clinicians based in Australia (69%) and Aotearoa New Zealand, which examined current clinical practice with regard to insulin regimen for children newly diagnosed with T1D, demonstrated a lack of consensus regarding starting regimen and dosing.[8] It was found that the implementation of an intensive regimen from diagnosis was less commonly opted for in Aotearoa New Zealand.

In July 2018, Christchurch Hospital implemented a protocol for intensive management of newly diagnosed T1D children <16 years. Prior to this no protocol existed, which resulted in differing approaches for insulin regimen, and the majority of those newly diagnosed were discharged on a twice daily insulin regimen. This protocol included carbohydrate counting from first meal, limiting snacks, flexible insulin dosing from first meal on subcutaneous insulin, avoidance of twice daily insulin regimen, targeted blood glucose levels from 4–8mmol/L irrespective of time of day, and corrections of blood glucose >12mmol/L while on injections between meals. The use of continuous glucose monitoring (CGM) technology was promoted. This study aimed to report the impact of the intensive management protocol at diagnosis on glycaemic control and management strategies 12 months after diagnosis compared to a historical cohort.

Methods

All newly diagnosed patients living in the Christchurch and West Coast Districts aged under 16 years old with T1D were treated in accordance with the new intensive-management protocol from 1 July 2018. T1D was defined as per the International Society of Pediatric and Adolescent Diabetes consensus statement.[9] The protocol consisted of a) carbohydrate counting from the first meal post-diagnosis, b) flexible subcutaneous insulin dosing from first meal, c) multi-daily injection (MDI) regimen d) targeted blood glucose levels from 4–8mmol/L irrespective of time of day, and e) corrections of blood glucose >12mmol/L (see Appendix 1).

Prior to and immediately after implementing the protocol, in-services for the ward staff were regularly given at nursing handovers to ensure the change in management was widely known and understood. This was to ensure that all healthcare professionals involved with newly diagnosed children and adolescents with diabetes delivered a consistent message.

This retrospective analysis from a prospectively recorded database analysed the first 70 consecutive young people diagnosed post-protocol implementation (enrolled from 1 July 2018 to 8 November 2020) and compared their HbA_{1c} at 12 months with the equivalent data from the last 70 consecutive people diagnosed immediately prior to the introduction of the new intensive-management protocol (diagnosed between 21 December 2015 and June 30 2018). Data that were collected for both groups included demographic data (age at diagnosis and prioritised ethnicity), presence and severity of diabetic ketoacidosis (DKA) at diagnosis, initial hospital stay duration, insulin regimen at 12 months post-diagnosis, use of CGM at 12 months and HbA_{1c} at 12 months post-diagnosis.

The MDI regimen was made up of a regular daily basal dose of long-acting insulin (glargine) together with multiple daily injections of rapid-acting insulin (insulin aspart) calculated from the patient's carbohydrate intake. The starting basal long-acting insulin dose was calculated using a starting value of 0.5x0.75–1.0U/kg. The rapid-acting doses were calculated in accordance with the practice of "carbohydrate counting", which combines standard carbohydrate to insulin ratios (CHO ratios) (calculated initially using the "500 rule", i.e., divide 500 by the total daily insulin dose to find the amount of carbohydrates in grams that 1 unit of rapid-acting insulin will cover). The CHO ratio calculation was adjusted for toddlers (<5 years) using 250 as the numerator rather than 500.[10–12] Insulin sensitivity factor (ISF) was defined using the 100 rule (divide 100 by the total daily insulin dose [0.75–1.0U/kg]). All calculations were reviewed daily by the inpatient care team, and then daily following discharge until glucose stability as per discretion of the diabetes educators.

In order for dosing ratios and carbohydrate counting to be implemented for inpatients, a new fully carbohydrate-counted menu was developed through the in-house catering service at Christchurch Hospital. This menu featured standard hospital breakfast and hot dinner options, while lunch was modelled on a "lunch box" with options for sandwiches, fruit, yoghurt and snacks. Between meals, snacks were reduced from 3 times daily to 2 times daily, eliminating a supper snack. Morning and afternoon tea snacks were further limited to <15g carbohydrates. For all food provided, the total amount of carbohydrates was calculated and declared on the menu to allow families to accurately begin carbohydrate counting. An important consideration was to advise families against providing extra food between these times.

Education took place over the inpatient admission, with a number of modules delivered by diabetes nurse educators and dieticians. As well as education regarding insulin administration, carbohydrate counting and carbohydrate-free foods, these sessions promoted CGM and the benefits of its

being initiated prior to discharge. Because CGM is not funded in Aotearoa New Zealand, social workers were closely involved and an application for the Child Disability Allowance was completed for each patient, which partially offset the cost of accessing CGM.

Following discharge, apart from daily phone contact, newly diagnosed families were seen in-clinic 2 weeks after diagnosis, and again 1 month later before entering into the routine 3-monthly follow-up.

In comparison, prior to the implementation of the protocol, there were inconsistent approaches at diagnosis. Specifically, insulin regimen was chosen *ad hoc*, there was no carbohydrate counting, hospital-provided meals were not carbohydrate counted, messaging on glucose targets was variable and promotion of CGM was inconsistent. However, all other educational modules were unchanged, as was the follow-up frequency after discharge.

The audit activity of this study was covered by "Clinical benchmarking utilising data from New Zealand Diabetes Centre Patient Management Systems"; Ethics Committee reference number HD18/098. Patient data are collected under a waiver of consent. Data collection was supported from a research grant provided by the Canterbury Medical Research Foundation.

A total of 70 in each cohort provided over 80% power to detect a moderate effect size of 0.5 with a two-sided alpha of 0.05. Assuming a HbA_{1c} standard deviation of 20mmol/mol[3] R, this would be a difference in HbA_{1c} of 10 mmol/mol between the two cohorts. Cohort characteristics were summarised by treatment group as counts (percentages) for categorical variables and as means and standard deviations (SD) or medians and interquartile ranges (IQR) for continuous normally or skewed variables respectively. Differences between groups were initially assessed using unadjusted tests (Student unpaired *t*-Test for HbA_{1c} and Pearson's Chi-squared test for categorical outcomes). Next, linear regression was used to estimate group differences in HbA_{1c} while firstly adjusting for the potential baseline confounders of non-European ethnicity and DKA at diagnosis, and secondly investigating the relative importance of use of CGM and insulin regimen at 12 months as effect modifiers.

Results

Table 1 describes the demographics of the two consecutive cohorts. The two cohorts were similarly matched, except for the post-intervention group being slightly older, a higher proportion having Māori ethnicity and a higher proportion presenting with DKA. All of the second cohort were educated with the intensive-management protocol, without exception.

Table 2 shows the 12-month data post-diagnosis of the two cohorts; the post-intensive-management cohort had an improved mean HbA_{1c} of 58.2±15.3mmol/L at 12 months, compared to 63.7±10.7mmol/mol in the historical group (p=0.014). As expected, there were notable differences in management modalities at 12 months post-diagnosis between the two cohorts, with near elimination of the twice daily insulin regimen—this being replaced by multi-daily injections—and an increased uptake of CGM (75% in cohort 2 vs 57% in cohort 1).

In order to assess which variables contributed to this improvement in HbA_{1c}, further analyses were undertaken. Firstly, we adjusted for baseline characteristics (non-European ethnicity and DKA at diagnoses were assumed as a predictor for higher HbA_{1c}). This showed the mean (95% confidence interval [CI]) difference between the two groups was now 7.3mmol/mol (95% CI 3.2–11, p<0.001), favouring the second cohort, which suggests that the changes in management more than overcame the predictive association of a poorer HbA_{1c} by ethnicity and DKA at diagnosis. We then adjusted for CGM use. This showed the mean (95% CI) difference between cohorts was 5.6 mmol/L (95% CI 1.5–9.6, p=0.007), favouring the second cohort, suggesting that the increased proportion of CGM use in cohort 2 had some impact on the overall difference, but did not explain all the difference. Similarly, adjusting for insulin regimen, the mean (95% CI) difference was 6.6 mmol/mol (95% CI 1.5–12, p=0.012) favouring cohort 2. These sequential analyses controlling for variables expected to predict outcome suggest the differences observed between two cohorts is multifactorial and not principally explained by either increased CGM use or insulin regime alone.

Discussion

Implementation of an intensive-management protocol from diagnosis in the management of children with T1D resulted in improved HbA_{1c} levels. This finding is not unexpected, with simi-

Table 1: Baseline characteristics of the two cohorts.

Characteristics	Cohort 1: pre-guidelines, n=70	Cohort 2: intensive, n=70
Gender	46% male	54% male
Median (IQR) age at diagnosis	9 (5–11) years	10 (8–12) years
Number (%) by prioritised ethnicity	NZ European, 63 (90%) Māori, 2 (3%) Pacific, 3 (4%) Other, 8 (11%)	NZ European, 56 (80%) Māori, 9 (13%) Pacific, 3 (4%) Other, 4 (6%)
Mean (SD) days in hospital during initial stay	3.3±1.3 days	3.3± 1.4 days
Mean (SD) number of clinics attended in first 12 months	5.7±1.2 clinics	5.5±1.0 clinics
Number (%) presenting with DKA† at diagnosis	23 (33%) 11 (16%) = severe‡ 10 (7%) = moderate‡ 1 (1%) = mild‡	28 (40%) 14 (20 %) = severe 11 (16%) = moderate 3 (4%) = mild

SD = standard deviation; DKA† = diabetic ketoacidosis; DKA‡ severity defined as severe if pH <7.0, moderate if 7.00–7.24 and mild if 7.25–7.30.

Table 2: Cohort comparison.

	Cohort 1: pre-guidelines, n=70	Cohort 2: intensive, n=70	
Number (%) by insulin regimen at 12 months	BD* 46 (66%) MDI† 16 (23%) CSII‡ 8 (11%)	BD 8 (11%) MDI 58 (83%) CSII 4 (6%)	p<0.001
Number (%) by use of rtCGM§ or isCGM§§ use at 12 months	40 (57%)	53 (75%)	p=0.020
Mean (SD) HbA_{1c} 12 months post-diagnosis	63.7±10.7 mmol/mol	58.3±15.3mmol/mol	p=0.014
Number (%) meeting target HbA_{1c} <53mmol/mol at 12 months	9 (13%)	31 (44%)	p<0.001

BD* = twice daily insulin regimen; MDI† = multi-daily injection insulin regimen; CSII‡ = insulin pump therapy; rtCGM§ = real-time continuous glucose monitoring; isCGM§§ = intermittently scanned continuous glucose monitoring.

lar results observed at the John Hunter Children's Hospital in Australia.[13] Further, international best-practice guidelines endorse intensive management from diagnosis.[2] Our experience demonstrates that translating this evidence-based approach is possible and effective.

Central to the change in practice was consistent messaging for the families coming from the whole team of healthcare professionals. Previous research has highlighted this is an important factor in influencing the success of management for adolescents.[14–16] For example, Swift et al. showed that adolescents tend to achieve lower HbA_{1c} targets at centres where there is a greater degree of agreement between health professionals in regard to these targets.[13] With the implementation of this protocol, a concerted effort was made to ensure we had a coordinated multidisciplinary team. Multiple ward in-services occurred both before and after implementation of the protocol in order to embed the change in practice. Consistent education was a key element of this messaging and, under this protocol, took place over the patients' initial admission at diagnosis. This inpatient model of education and information dissemination capitalises on the opportunity presented while patients and whānau (family) are present, engaged and have time to take information on board. Families were provided information about glucose targets, insulin dosing (insulin action where rapid-acting insulin is calculated according to carbohydrate intake and correction factors, importance of 15 minute pre-bolus), carbohydrate counting and the practicalities of administering insulin (for example, injection technique) prior to discharge. The multidisciplinary team (diabetes educator, endocrinologist and dietitian) are available at all subsequent outpatient appointments, and therefore there exists a system for ongoing education and reinforcement of management goals.

It is important to note that diabetes management is currently going through rapid evolution. With higher use of real-time CGM,[15] and with modern automated insulin delivery systems becoming increasingly prevalent within 12 months of diagnosis, there is great potential for even further improvements to be seen in the future. For example, Prahalad et al. showed that CGM from diagnosis results in sustained improved HbA_{1c}.[17] While our cohort had quite high rates of CGM use, most were using intermittently scanned rather than real-time CGM due to cost. It remains important for diabetes clinics in Aotearoa New Zealand to prepare for future improved access to these technologies and rapidly translate this to routine care as soon as possible in the patient journey from diagnosis.

Limitations of this study were that it was an audit, with a retrospective control arm, as opposed to a randomised control trial comparing the two interventions. Thus, is it possible that there are additional factors interacting to influence the results, especially as we were not able to delineate between real-time and intermittently scanned CGM, or the proportion in either cohort that had CGM applied at initial diagnoses (but can safely be assumed was higher in the second cohort) or total daily dose. The improved HbA_{1c} demonstrated is likely to reflect multiple factors, broadly reflecting adjusted education, insulin injection regimen, improving diabetes technology and consistent messaging from the clinical service. The second cohort was affected by COVID-19 lockdowns and some clinic appointments were made by video or telephone, and the impact of this has not been analysed. It should be highlighted that the cohort in the study was predominantly European, and results may not be generalisable to centres with different ethnic makeups. A strength of this study was the ability to collect a full dataset for each patient involved in the study thanks to the routine collection of data at admission and follow-up of patients who are diagnosed with T1D in Christchurch.

In conclusion, this study provides evidence to support the efficacy of intensive management from diagnosis for children with T1D and could be used as a model for other centres in Aotearoa New Zealand who are yet to deploy this evidence-based practice.

COMPETING INTERESTS
None to declare.

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REFERENCES
1. Nathan DM, Genuth S, Lachin J, et al. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med. 1993;329(14):977-86. doi: 10.1056/NEJM199309303291401.
2. de Bock M, Codner E, Craig ME, et al. ISPAD Clinical Practice Consensus Guidelines 2022: Glycemic targets and glucose monitoring for children, adolescents, and young people with diabetes. Pediatr Diabetes. 2022;23(8):1270-6. doi: 10.1111/pedi.13455.
3. James S, Perry L, Lowe J, et al. Suboptimal glycemic control in adolescents and young adults with type 1 diabetes from 2011 to 2020 across Australia and New Zealand: Data from the Australasian Diabetes Data Network registry. Pediatr Diabetes. 2022;23(6):736-41. doi: 10.1111/pedi.13364.
4. Phelan H, Clapin H, Bruns L, et al. The Australasian Diabetes Data Network: first national audit of children and adolescents with type 1 diabetes. Med J Aust. 2017;206(3):121-5. doi: 10.5694/mja16.00737.
5. Prahalad P, Yang J, Scheinker D, et al. Hemoglobin A1c Trajectory in Pediatric Patients with Newly Diagnosed Type 1 Diabetes. Diabetes Technol Ther. 2019;21(8):456-61. doi: 10.1089/dia.2019.0065.
6. Cengiz E, Connor CG, Ruedy KJ, et al. Pediatric diabetes consortium T1D New Onset (NeOn) study: clinical outcomes during the first year following diagnosis. Pediatr Diabetes. 2014;15(4):287-93. doi: 10.1111/pedi.12068.
7. Nirantharakumar K, Mohammed N, Toulis KA, et al. Clinically meaningful and lasting HbA_{1c} improvement rarely occurs after 5 years of type 1 diabetes: an argument for early, targeted and aggressive intervention following diagnosis. Diabetologia. 2018;61(5):1064-70. doi: 10.1007/s00125-018-4574-6.
8. Selvakumar D, Al-Sallami HS, de Bock M, et al. Insulin regimens for newly diagnosed children with type 1 diabetes mellitus in Australia and New Zealand: A survey of current practice. J Paediatr Child Health. 2017;53(12):1208-14. doi: 10.1111/jpc.13631.
9. Libman I, Haynes A, Lyons S, et al. ISPAD Clinical Practice Consensus Guidelines 2022: Definition, epidemiology, and classification of diabetes in children and adolescents." Pediatr Diabetes. 2022 Dec;23(8):1160-1174. doi: 10.1111/pedi.13454.
10. Enander R, Gundevall C, Strömgren A, et al. Carbohydrate counting with a bolus calculator improves post-prandial blood glucose levels in children and adolescents with type 1 diabetes using insulin pumps. Pediatr Diabetes. 2012;13(7):545-51. doi: 10.1111/j.1399-5448.2012.00883.x.
11. Cengiz E, Danne T, Ahmad T, et al. ISPAD Clinical Practice Consensus Guidelines 2022: Insulin treatment in children and adolescents with diabetes. Pediatr Diabetes. 2022;23(8):1277-96. doi: 10.1111/pedi.13442.
12. Davidson PC, Hebblewhite HR, Steed RD, Bode BW. Analysis of guidelines for basal-bolus insulin dosing: basal insulin, correction factor, and carbohydrate-to-insulin ratio. Endocr Pract. 2008;14(9):1095-101. doi: 10.4158/EP.14.9.1095.
13. Phelan H, King B, Anderson D, et al. Young children with type 1 diabetes can achieve glycemic targets without hypoglycemia: Results of a novel intensive diabetes management program. Pediatr Diabetes.

2018;19(4):769-75. doi: 10.1111/pedi.12644.
14. Swift P, Skinner TC, de Beaufort CE, et al. Target setting in intensive insulin management is associated with metabolic control: the Hvidoere Childhood Diabetes Study Group Centre Differences Study 2005. Pediatr Diabetes. 2010;11(4):271-8. https://doi.org/10.1111/j.1399-5448.2009.00596.x.
15. Skinner TC, Lange KS, Hoey H, et al. Targets and teamwork: Understanding differences in pediatric diabetes centers treatment outcomes. Pediatr Diabetes. 2018;19(3):559-65. doi: 10.1111/pedi.12606.
16. Johnson SR, Holmes-Walker DJ, Chee M, et al. Universal Subsidized Continuous Glucose Monitoring Funding for Young People With Type 1 Diabetes: Uptake and Outcomes Over 2 Years, a Population-Based Study. Diabetes Care. 2022 Feb 1;45(2):391-397. doi: 10.2337/dc21-1666.
17. Prahalad P, Zaharieva DP, Addala A, et al. Improving Clinical Outcomes in Newly Diagnosed Pediatric Type 1 Diabetes: Teamwork, Targets, Technology, and Tight Control-The 4T Study. Front Endocrinol (Lausanne). 2020;11:360-360. doi: 10.3389/fendo.2020.00360.

Appendix 1

Hospital HealthPathways Waitaha | Canterbury

MedChart

Inpatient Management of Diabetes in Children

This pathway is about the inpatient management of patients with type 1 diabetes during their initial presentation (including mild diabetic ketoacidosis able to be managed with subcutaneous insulin), subsequent admissions for diabetes, or admissions for another condition. It is not about management of diabetic ketoacidosis requiring an insulin infusion, or the outpatient management of diabetes. See also Diabetic Ketoacidosis (DKA) in Children.

Background

About inpatient management of diabetes in children

About inpatient management of diabetes in children

Good glycaemic control from diagnosis predicts long-term blood glucose control, and may preserve beta-cell mass and prolong the honeymoon period. Consistent management and messaging from all staff involved in looking after the patient helps to achieve this.

Assessment

1. Ensure the patient is not in diabetic ketoacidosis as, if present, this needs to have resolved before subcutaneous insulin is started.
2. Check the patient's blood glucose level to assess for:
   - hypoglycaemia (blood glucose level less than 4.0 mmol/L) or
   - hyperglycaemia (blood glucose level greater than 12.0 mmol/L).

Management

1. Determine insulin dose:
   - Consider factors which can influence the patient's insulin requirement .

     Factors which can influence the patient's insulin requirement

     - Age
     - Body mass index
     - Presence of ketones
     - How early the patient presented

   - Determine the total daily insulin dose to use as a starting point .

     Total daily insulin dose to use as a starting point

     The starting point for most patients is 1 unit/kg/day. Any variation from this dose must be discussed with the senior medical officer (SMO) responsible for the patient (some children who are lean, newly diagnosed and young may only require 0.5 units/kg/day).

     When calculating the insulin dose:

     - Round down to the nearest whole unit of insulin for all patients aged 5 years or older.
     - Consider half units for patients younger than 5 years, with an insulin requirement less than 15 units/day.

2. Determine detail of the insulin dosing regime.
   - Use a multi-daily-injection (MDI) insulin regime .

     Multi-daily-injection (MDI) insulin regime

MDI insulin regimens are the best way of achieving optimal glycaemic control.

MDI insulin regimens involve a basal dose of Lantus, with fast acting insulin (Novorapid or Humalog) injected before meal times and to correct hyperglycaemia. All children should be started on this regimen at diagnosis.

- Calculate the Lantus basal insulin dose as 50% of the total daily insulin dose. Round down to the nearest half a unit.
- Prepare the following information which will be required to ⌄ determine the fast-acting insulin (Novorapid/Humalog) dosing :

Determine the fast acting insulin (Novorapid/Humalog) doses

The insulin dose is calculated as a combination of the carbohydrate ratio (the amount of insulin for the carbohydrate in the meal) and a correction for any hyperglycaemia (the amount of insulin required to return the glucose level back to target, known as insulin sensitivity factor). Avoid using the term sliding scale to patients, because it does not correspond to our education goal of learning to use carbohydrate ratios and insulin sensitivity.

  - ⌄ Carbohydrate ratio , which is used to cover the carbohydrate consumed.

Carbohydrate ratio

- The carbohydrate ratio specifies how many grams of carbohydrate a single unit of insulin will cover in a meal. For example, for a child with a carbohydrate ratio of 1 to 10, one unit of insulin will cover 10 g of carbohydrate.
- The carbohydrate ratio is initially calculated as 1 unit of insulin for x grams of carbohydrate, where $x = 500 \div$ total daily insulin dose. This is conservative, and will often need to be made more aggressive. While in hospital, children with type 1 diabetes will have a menu provided with carbohydrate amounts.

  Case example – A 50 kg child with total daily insulin requirements of 1 unit/kg/day requires 50 units/day. The carbohydrate ratio is then 1 to 10 (1 unit of insulin for every $500 \div 50 = 10$ g of carbohydrate). Therefore if the child eats a 50 g carbohydrate meal, they will need to inject 5 units of insulin to cover this.

- Children aged younger than 4 years need a carbohydrate ratio that is stronger with respect to their weight. They will often need it calculated as 250 ÷ total daily dose. Discuss with the endocrinologist.

  Case example – A 10 kg child with total daily insulin requirements of 1 unit/kg/day requires 10 units/day. The carbohydrate ratio is then 1 to 25 (1 unit for every 250 ÷ 10 = 25 g of carbohydrate). Therefore if the child eats a 50 g carbohydrate meal, they will need to inject 2 units of insulin to cover this.

- Insulin sensitivity factor (ISF) , which is used to correct hyperglycaemia.

Insulin sensitivity factor

The insulin sensitivity factor is used for correcting hyperglycaemia. It specifies by how much 1 unit of insulin will decrease the blood glucose level in mmol/L.

The insulin sensitivity factor is calculated as 1 unit to reduce the blood glucose by x mmol/L, where x = 100 ÷ total daily dose.

Case example – A 50 kg child, with total daily insulin requirements 1 unit/kg/day requires 50 units/day. The insulin sensitivity factor is 1 in 100 ÷ 50 = 1 to 2 (1 unit will reduce the glucose level by 2 mmol/L within 3 hours). The glucose target is 6 mmol/L irrespective of the time of day or night. Therefore if the child has a blood glucose level of 12 mmol/L, they will need to inject 3 units of insulin to bring their blood glucose level back down to 6 mmol/L.

- Document both values in the Cortex, using the Paediatric Diabetes Clinical Summary.
- Generate a personalised insulin dose grid . Recalculate this grid daily, based on feedback from the Diabetes Team (Nurse Educators or Endocrinologists).

Generate a personalised insulin dose grid

Open the spreadsheet used to calculate personalised insulin doses and follow the instruction on the spreadsheet for calculating, printing, and saving the grid. For children aged 4 years and over, use the full unit grid , and for children aged younger than 4 years, use the half unit grid .

> For the first grid of the admission, use the carbohydrate ratio and insulin sensitivity factor based on the instructions above. The target blood glucose level is always 6 mmol/L. The first grid is a starting point, and subsequent grids may be adjusted depending on the patient's blood glucose profile.
>
> 1. Save a copy of the grid by using "Save as" and making the file name *Lastname_Firstname_NHI_DD.MM.YR* and saving to the location *G:\Division\PAE\COMMON\Diabetes\Medchart\Patients*
> 2. After the grid is printed add a patient label, add your name and signature, and the date, and place in the front of the patient's notes. Discard any previous grids.

3. Prescribe insulin:
   - Prescribing Lantus:
     - Prescribe Lantus before dinner at 5.30 pm.
     - The first dose can be given any time between pre-dinner and midnight. However, if the patient finishes their insulin infusion before breakfast, give half the calculated Lantus dose with breakfast, and the full calculated Lantus dose pre-dinner that evening.
   - Prescribing Novorapid/Humalog for meals

> **Prescribing Novorapid/Humalog for meals**
>
> - Make sure there is an up-to-date personalised insulin dose grid
> - In Medchart:
>   - Choose the insulin to be administered (Novorapid or Humalog) .
>
> > **Choose the insulin to be administered (Novorapid or Humalog)**
> >
> > These are equivalent. Choose based on what is available on the ward (usually Novorapid), or what the patient takes at home if they have brought their own insulin to hospital.
>
>   - Chart a dose range, which should be from 0 to the highest insulin dose on the patient's personalised insulin dose grid.

- Prescribe as a scheduled medicine and choose the mealtime the insulin is for from the dropdown box labelled Schedule. There is no option for afternoon tea, so pick once daily and write Afternoon Tea in the Qualifier box.
- Record the current carbohydrate ratio and the insulin sensitivity factor in the qualifier box.
- Make sure there is a separate prescription for each of breakfast, lunch, afternoon tea, and dinner.
- For the afternoon tea dosing, use the carbohydrate ratio only, i.e. use the top line of the grid to determine what insulin dose range to chart.

- Prescribing Novorapid/Humalog for corrections

Prescribing Novorapid/Humalog for corrections

- Don't give additional corrections that are not part of pre-meal insulin unless the last Novorapid/Humalog dose administered was at least 3 hours beforehand.
- In Medchart:
  - Choose the insulin to be administered (Novorapid or Humalog) .
  - Chart a dose range, which should be from 0 to the highest insulin dose in the zero carbohydrate column of the patient's personalised insulin dose grid. Check that the target blood glucose level on the grid is 6 mmol/L.
  - Prescribe as an as required (PRN) medicine.
  - Record the current insulin sensitivity factor in the qualifier box.
  - The clinical nurse specialist (CNS) or registrar must confirm the correction dose, according to the insulin sensitivity factor (ISF) that has been prescribed.

4. Administer insulin at mealtimes:
   - Calculate the insulin dose based on the amount of carbohydrates that will be consumed and the pre-meal blood glucose, involve the family and make reference to the patient's personalised insulin dose grid.

Amount of carbohydrates that will be consumed

> As all food should be carbohydrate counted, the kitchen will provide specifically designed day meals and snacks, labelled with carbohydrate amount. Dinner meals will be delivered according to the normal menu.
>
> Advise families not to provide extra food between meals, as it will not be covered appropriately with the insulin prescribed.

- Give short-acting insulin (Novorapid/Humalog) 15 to 30 minutes before the meal.
- Use an insulin pen to administer all subcutaneous insulin. Avoid insulin syringes and mixing different insulins. 6 mm and 8 mm pen needles are suitable for most children and adolescents respectively.

5. Advise the patient and family on food intake .

> **Advise the patient and family on food intake**
>
> Ensure patients with diabetes:
>
> - have three main meals.
> - limit snacks between main meals. Morning tea and afternoon tea should contain less than 15 g of carbohydrate. Older adolescents may be an exception to this at afternoon tea. An extra insulin dose will be required if more than 15 g of carbohydrate is eaten at that point.
> - avoid supper (i.e., no further meals after dinner, and snacks must be free from carbohydrate). There is no insulin prescribed to cover late night food, and will result in overnight hyperglycaemia.
> - strictly avoid all sugar-containing carbonated drinks, fruit juice, and lollies. Potato chips and other high fat snacks should be highly discouraged.
>
> Ensure all food intake is recorded by the nurse on the fluid balance chart, documenting the carbohydrate intake.

6. Monitor blood glucose – The blood glucose target range for any patient with type 1 diabetes is 4.0 to 8.0 mmol/L:
   - If the patient has a continuous glucose monitor and the blood glucose level is 4 to 15 mmol/L, use this for blood glucose monitoring. Otherwise, take a capillary blood glucose measurement by finger prick.
   - Once out of diabetic ketoacidosis (DKA), and on regular subcutaneous insulin, monitor glucose in all patients at the following times:

- Before breakfast
- Three hours after breakfast time rapid acting insulin
- Lunch
- Three hours after lunchtime rapid acting insulin
- Before dinner
- Before bed (9.00 pm at the latest)
- Midnight
- 4.00 am
- At any time the patient is symptomatic for hypoglycaemia.

7. Monitor blood ketones:
   - At admission and then whenever measuring blood glucose until ketone level less than 0.5 mmol/L for more than 6 hours. This information is used by the diabetes team to determine whether any changes to overall diabetes management are required.
   - Whenever blood glucose level is greater or equal to 12 mmol/L on two consecutive readings taken at least 3 hours apart.
8. Manage hypoglycaemia and hyperglycaemia .

Manage hypoglycaemia and hyperglycaemia

Hypoglycaemia – Treat hypoglycaemia (BSL less than 4.0 mmol/L) according to the Hypoglycaemia in Children pathway.

Hyperglycaemia:

- Blood glucose level greater than 12 mmol/L:
  - Daytime – Use the patient's personalised insulin grid to correct at main mealtimes.
  - Night-time – Give a correction using the short-acting insulin that has been prescribed for as required administration, but only if the last short-acting insulin was given more than 3 hours ago. Use the zero carbohydrate column on the personalised insulin grid to determine how much insulin to give according to the blood glucose level.

- Blood glucose level greater than or equal to 12 mmol/L on 2 consecutive readings taken 3 hours apart – Check ketones:
  - If blood ketone level is less than 1 mmol/L, manage as for blood glucose level greater than 12 mmol/L.
  - If blood ketone level is greater or equal to 1 mmol/L, manage as for blood glucose level greater than 12 mmol/L but give 1.5 times the calculated correction dose of insulin. Optimise fluid intake as this can help clear ketones.
- If you are unsure about how much insulin to administer, seek advice from a senior colleague or registrar.

9. Make arrangements for discharge:
   - Ensure diabetes education, if required, has taken place.
   - Request follow-up as directed by endocrinology using the Cortex Paediatric Follow-up Order.

   Prescription of outpatient medications, where required, will be done by the diabetes team.

PARALLEL PAGES

Diabetes Diagnosis in Children

SOURCES

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