**Running title page**

Antecedents of DKA

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**Title page**

# Pathways to reduce diabetic ketoacidosis with new onset type one diabetes: evidence from a regional pediatric diabetes center: Auckland, New Zealand, 2010 to 2014.

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**Abstract**

*Background:* There has been little change in the incidence of diabetic ketoacidosis (DKA) in newly diagnosed type 1 diabetes mellitus (T1DM) in children and adolescents in most developed countries.

*Objectives:* To assess potentially modifiable antecedents of DKA in children < 15 years of age with new onset T1DM.

*Methods*: Retrospective review of prospectively collected data from a complete regional cohort of children with T1DM in Auckland (New Zealand) from 2010 to 2014. DKA and severity were defined according to the ISPAD 2014 guidelines.

*Results:* 263 children presented with new onset T1DM during the 5 year study period at 9.0 years of age [range 1.0-14.7], of whom 61% were NZ-European, 14% Maori, 13% Pacifica and 11% other. 71 patients (27%) were in DKA, including 31 mild, 20 moderate, and 20 severe DKA. DKA was associated with no family history of T1DM, higher HbA1c values at presentation, self-presenting to secondary care, health care professional contacts in the 4 weeks before final presentation, and greater deprivation. Although a delay in referral from primary care for laboratory testing was common (81/216), only delay for more than 48 hours was associated with increased risk of DKA (11/22 > 48 h *vs*. 12/59 patients referred at < 48 hours, P = 0.013).

*Conclusions:* These data suggest that in addition to lack of family awareness potentially modifiable risk factors for new onset DKA include prolonged delay for laboratory testing and a low index of medical suspicion for T1DM leading to delayed diagnosis.

**Key words (5)**

T1DM; Insulin; Diabetic Ketoacidosis; Children

**Abbreviations**

| | |
|---|---|
| BMI | Body Mass Index |
| DKA | Diabetic Ketoacidosis |
| HCP | Health care professional |
| SDS | Standard Deviation score |
| SES | Socio-Economic-Status |
| T1DM | Type 1 Diabetes Mellitus |

## Introduction

Diabetic ketoacidosis (DKA) is the leading cause of serious morbidity in children with type 1 diabetes mellitus (T1DM) (1-3). The diagnosis of T1DM is preceded by a variable period of symptoms including weight loss, polyuria and polydipsia (2). Early insulin replacement prevents DKA. Thus the presence of DKA is largely a consequence of delay in diagnosis, and in turn, delayed initiation of insulin therapy (4). Although, screening for children at high risk for T1DM is possible and can reduce the risk of DKA, it is unlikely to be economically viable to screen the whole population (5).

Greater rates of DKA in patients with newly diagnosed T1DM have been associated with income inequality, delay in diagnosis, minority ethnicity, delayed access to healthcare, older age (except for toddlers), and absence of a first degree relative with T1DM, as recently reviewed (1). These associations with DKA at diagnosis of T1DM support the hypothesis that there is a lack of awareness of the symptoms of T1DM by parents and physicians, and thus that there is scope for more timely intervention.

There is some evidence that delayed medical diagnosis of T1DM can be one factor leading to greater risk of DKA, although there are conflicting results (4). For example, in Ontario, Canada, children with new onset diabetes presenting with DKA were more likely to have had a healthcare professional (HCP) encounter before diagnosis, but were less likely to have had relevant laboratory tests compared to those without DKA (6). Similarly, in Southampton, UK, patients with new onset T1DM who had multiple HCP visits before diagnosis had lower pH on admission (7). In addition to missed diagnoses, in some cases secondary referral may have been delayed by awaiting the results of non-urgent investigations such as fasting blood sugars (7, 8). Interestingly, a nationwide study in France of 1299 children < 15 years with T1DM found that severe

DKA was associated with self-presentation to hospital compared with physician referral (26.6% vs. 7.6%), and reduced risk in children with a family history of T1DM (9). These findings strongly suggest that both medical and family awareness of T1DM affect the risk of DKA at presentation.

New Zealand has a social security system that provides free medical care (insurance is not required), and care is provided in the community through primary care delivered by general practitioners. As a result, income is not a direct impediment for take-up of medical care, and the associated costs of T1DM to patients' families are minimal. We recently reported that although the incidence of T1DM increased over 15 years in Auckland, New Zealand, in line with worldwide trends (10), there had been no change in the rate of DKA associated with new onset T1DM (11). These findings suggest that there is scope to further improve diagnosis of T1DM before DKA develops.

Thus, the aim of this study was to investigate potentially modifiable factors associated with DKA in children aged <15 years with new onset T1DM. We examined the role of previous HCP contacts before diagnosis, referral from primary care compared to self-presentation to hospital, and whether the impact was modified by a family history of T1DM in first degree relatives, by ethnic background or by deprivation.

## Methods

Ethics approval was granted by the Auckland DHB Research Review Committee, study number: A+ 6827. All procedures followed were in accordance with the ethical standards of this committee. All patients provided written consent prior to being entered into the Starbase (the Starship Children's Hospital Diabetes Database). Written or verbal informed consent was not required, as this study involved an audit of routine clinical practice.

### Participants

A retrospective audit was undertaken of all patients <15 years of age presenting to or transferred to Starship Children's Hospital from January 1st 2010 to Dec 31st 2014 inclusive. These patients were identified from Starbase. Further information was obtained from the clinical record as required. Patients were excluded from this study if they did not have T1DM, were >15 years of age, or if admission for management of newly diagnosed T1DM did not occur in Auckland.

T1DM was diagnosed based on clinical features. All patients had elevated blood glucose at presentation (a random measurement of >11.1 mmol/l and/or fasting blood glucose >7.1 mmol/l) and presented with classical symptoms. In addition, all patients met at least one of the following criteria: a) DKA; b) presence of pre-T1DM associated antibodies (glutamic acid decarboxylase, islet antigen 2, islet cell, or insulin autoantibodies); or c) on-going requirement for insulin therapy. Subjects with type 2 diabetes, monogenic or other forms of diabetes (e.g. cystic fibrosis related) were excluded from this study.

### Study Parameters

Demographic data (including age, sex and ethnicity), weight and height during the admission, and laboratory test results were tabulated. DKA was defined according to ISPAD 2014 guidelines as the combination of ketosis, hyperglycemia and acidosis (venous pH < 7.3 or bicarbonate <15 mmol/l) (2). DKA was further classified as mild (venous pH < 7.3 or bicarbonate <15 mmol/l), moderate (pH < 7.2 or bicarbonate <10 mmol/l), or severe (pH < 7.1 or bicarbonate <5 mmol/l) (2).

*Definition of referral patterns*

Primary care referral was confirmed by examination of the electronic discharge summary, clinical records or referral letter if available. Primary care includes both general practice, and after-hours community care. The timing of referral from primary care to secondary care was established by review of the clinical notes and by identifying the date of any community blood tests compared to date and time of admission to hospital. Delay was categorized as < 24 hours, 24 to 48 hours or > 48 hours delay. Self-presentation was defined as attendance for emergency hospital care without a referral.

Family history of diabetes was defined as any first degree relative with T1DM. HCP contacts were defined as any contact with a HCP in the previous 4 weeks, not including a contact leading to referral to secondary care. Socioeconomic status was categorized using the New Zealand Index of Deprivation 2013 (NZDep2013), a geo-coded deprivation score derived from current residential address (12).

**Statistical analysis**

Frequencies were compared by Fishers exact test. Univariate and multivariate binary logistic regression were used to assess factors associated with DKA and recent HCP

contacts, using IBM SPSS v 22 (IL, USA). Data were considered significant when $P < 0.05$. Data are mean ± standard error of the mean (SEM).

## Results

In total 296 patients <15 years of age with diabetes were recorded in Starbase over the 5 year time period. 33 patients were excluded; 28 had T2DM, 3 had other forms of diabetes (pancreatectomy, cystic fibrosis-related diabetes and monogenic diabetes), and 2 were not residents of Auckland.

### DKA

The 263 patients were 9 years old [mean; range 1.0-14.7] at the time of diagnosis of T1DM. 71 patients (27%) were in DKA at the time of presentation (Table 1). Of these, 20 were severe, 20 moderate and 31 were mild. Patients with DKA had higher HbA1c values, were less likely to have a family history of T1DM and less likely to have been referred from primary care, but were more likely to have recent HCP contact, and had a greater index of deprivation. There was no significant univariate effect of age, sex, BMI or ethnicity. Although some delay to obtain laboratory tests was common before referral from primary care (81/216 patients, Table 1), overall, delay was not associated with risk of DKA (P = 0.43). However, there was a significant effect of the duration of delay, such that DKA occurred in 11/22 (50%) of patients for whom referral was delayed by > 48 hours after the first appointment, compared with 12/59 (20.3%) of patients referred within less than 48 hours (P = 0.013).

DKA was independently associated with lack of family history of T1DM (B 2.2, P = 0.006), recent HCP contacts (B 1.3, P = 0.003), greater deprivation (B 0.14, P = 0.009), greater age (B 0.14, P = 0.002), self-presentation (B 1.0, P = 0.019) and lower BMI (B 0.18, P = 0.002) in multivariate binary logistic analysis, excluding biochemical measures of DKA and diabetes.

**Referral patterns**

To better understand the effect of referral from primary care compared to self–presentation directly to secondary care at a hospital accident and emergency department we examined a detailed breakdown of patient referral status and family history (Table 2). This suggested that a much higher proportion (17/45, 38%) of patients who self-presented to the emergency department had a family history of T1DM, compared with those who were referred from primary care (15/216, 7%, $P < 0.001$). Very few patients with a family history of T1DM were in DKA, regardless of whether they were referred from primary care (1 patient, 6.7%) or self-presented (1 patient, 5.9%). By contrast, a much higher proportion of patients with no family history who self-presented were in moderate to severe DKA (46% of self-presentations vs 12% referred from primary care, $P < 0.001$).

**Prior Health Care Professional contacts in the 4 weeks before diagnosis**

33 patients had one or more HCP contacts in the 4 weeks before diagnosis to either general practice or a general practitioner-run after hours care center. These patients had a significantly increased rate of DKA (Table 3). Univariate analysis suggested that HCP contacts were associated with lower HbA1c at presentation, more likely to be non-European, and with a trend to greater deprivation ($P = 0.06$). There was no association with primary care referral, age, sex or BMI.

Multivariate binary logistic regression (excluding biochemical variables) suggested that recent HCP contacts were independently associated with non-European ethnicity (B 0.84, $P = 0.03$). There was no significant independent effect of primary care referral, age, sex, BMI or family history on recent HCP contact.

## Discussion

The majority of children and adolescents presenting with new onset T1DM over a 5 year interval in Auckland, New Zealand, were referred from primary care and were not in DKA. Referral from primary care and a family history of T1DM were associated with lower rates of DKA (1, 9). Nevertheless, despite the positive overall effect of referral from primary care, in some cases referral was delayed for over 48 hours. This single factor contributed at least 15% of all cases of DKA. Conversely, greater deprivation and, in patients with no family history, self-presentation to secondary care were associated with a particularly high risk of DKA. Finally, children with new onset T1DM who presented in DKA, particularly those of non-European ethnicity, were more likely to have had one or more contacts with medical professionals in the month before diagnosis, consistent with previous evidence that delayed medical diagnosis continues to contribute to risk of DKA (6). Preceding HCP contacts were associated with 24% (17/71) of cases of DKA; 2 patients had both HCP contacts and very delayed referral, thus these data suggest that 37% (26/71) of DKA at presentation could be potentially prevented by improved referral practice and a higher medical index of suspicion.

Patients in DKA at presentation had higher HbA1c values and, in multivariate analysis, lower BMI and older age (1). The combination of higher HbA1c and lower BMI is consistent with the concept that T1DM had been progressing for longer, leading to more prolonged exposure to hyperglycemia, and greater catabolism. Risk of DKA was independently increased with greater deprivation in our population, despite free medical care. Speculatively, this might be related to limited time to visit primary providers. Alternatively, lower affluence may be in part a marker of parental education or lack of self-confidence. Further studies are needed to assess how long

families had been aware of the symptoms of T1DM before presentation. Small studies suggest that families are aware of symptoms for some time before consultation with a HCP (8).

Reassuringly, referral from primary care was independently associated with reduced risk of DKA compared to self-presentation. In just over a third of patients referred from primary care, the referral was delayed to obtain laboratory confirmation rather than immediately referring to secondary care. Although all cases of suspected T1DM should be referred urgently for definitive diagnosis and treatment (2), in the present study the risk of DKA was only increased if the delay was greater than 48 hours. These results support the use of either urine spot tests or point of care glucometers to immediately test for hyperglycemia (7, 13), to enable rapid referral to secondary care. If this single factor could be completely eliminated, the present findings suggest that the rate of DKA could be reduced by up to 15%. Speculatively, it may be that primary care doctors are more used to managing adult T2DM, where initial treatment may not be urgent, and do not appreciate how rapidly DKA can develop in childhood T1DM. If so, targeted education and refined protocols may help reduce unnecessary delay.

A family history of T1DM in a first degree relative was highly and independently protective irrespective of the mode of presentation, consistent with considerable previous evidence (1, 9). The mechanism is very likely to be family awareness of the symptoms of T1DM, allowing timely diagnosis before DKA can develop. Indeed, the authors' experience is that many of these families had already documented an elevated blood sugar before presentation. The importance of family awareness is strengthened by the observation that more than half (61%) of patients with no family history who self-presented to emergency care were in DKA at the time of presentation, compared with just 26% of those who went through primary care. This

suggests that these families did not appreciate the significance of the initial signs of new T1DM and only sought medical care when their child became unwell.

A further major risk factor for DKA in the present study was HCP contact in the previous 4 weeks before referral from primary care or self-presentation. We do not know the reasons for these consultations. Nevertheless, the association with subsequent DKA infers that at least some of these contacts represented missed opportunities for diagnosis. This factor was associated with 24% cases of DKA, suggesting that an increased index of medical suspicion for T1DM would have potential to substantially further reduce the risk of DKA at presentation of T1DM. All children who present to the Emergency Department at Starship Hospital have a test for urine glucose. Potentially, point of care testing for urine or blood glucose, followed by ketone screening if the glucose level is elevated, in primary care might reduce the risk of delayed diagnosis (7, 13).

Interestingly, having recent HCP contacts was associated with non-European ethnicity. The reasons are unknown, but it is reasonable to consider whether in some cases delayed diagnoses might reflect culturally determined expectations or communication leading to inequality of use although not access to medical care. Alternatively, there is increased risk of T2DM in adolescent Maori and Pacifica patients in New Zealand (14). Potentially, although T2DM is rare below the age of 10 years, and the rates of T1DM in children are now similar between ethnic groups in New Zealand (15), it is possible that community and HCP focus on T2DM may contribute to a lack of appreciation of the risks of T1DM in children. These are important avenues for further investigation.

In conclusion, this study has highlighted that delayed referral from primary care for > 48 h and, at least potentially, missed diagnosis over the 4 weeks before presentation of

T1DM, are important modifiable antecedents of DKA, that together contributed to approximately 37% of cases of DKA. Both deprivation and minority ethnicity appear to further increase risk of DKA. Further research is important to understand how to address these issues in a culturally sensitive manner.

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Table 1. Demographic and biochemical features of children with new onset T1DM, comparing those who were not in DKA with those in DKA

| | All subjects | No DKA | DKA | P-value |
|---|---|---|---|---|
| N | 263 | 192 (73%) | 71 (27%) | |
| Age (y) | 9.0 (0.2) | 8.8 (0.3) | 9.6 (0.4) | 0.14 |
| Female | 123 (46.8%) | 84 (43.8%) | 39 (54.9%) | 0.11 |
| BMI $kg/m^2$ | 17.3 (0.3) | 17.7 (0.3) | 16.5 (0.4) | 0.06 |
| pH | 7.31 (0.01) | 7.38 (0.04) | 7.15 (0.01) | <0.001 |
| Bicarbonate (mmol/L) | 19.9 (0.4) | 22.8 (0.3) | 12.3 (0.71) | <0.001 |
| Glucose (mmol/L) | 27.0 (0.6) | 26.5 (0.7) | 28.1 (1.2) | 0.23 |
| HbA1c (mmol/mol) | 108.8 (1.8) | 105.3 (2.1) | 118.7 (2.8) | <0.001 |
| Ethnicity | | | | 0.074 |
| NZ European | 160 (60.8%) | 122 (63.5%) | 38 (53.5%) | |
| Maori | 38 (14.4%) | 29 (15.1%) | 9 (12.7%) | |
| Pacific Islander | 35 (13.3) | 23 (12.0%) | 12 (16.9) | |
| Other | 30 (11.4) | 18 (9.4%) | 12 (16.9%) | |
| Deprivation Score (range 1–10) | 4.8 (0.2) | 4.5 (2.9) | 5.6 (0.36) | 0.01 |
| Positive family history of T1DM | 33 (12.6%) | 31 (16.1%) | 2 (2.8%) | <0.001 |

| | | | | |
|---|---|---|---|---|
| HCP contacts in the previous 4 weeks* | 33 (12.5%) | 16 (8.3%) | 17/71 (23.9%) | 0.001 |
| Primary care referral # | 215 (82.3%) | 163 (85.8%) | 53 (74.6%) | 0.043 |
| Delay before referral from primary care# | 81/216 (37.7%) | 59/163 (36.2%) | 22/53 (41.5%) | 0.43 |

Data are mean (SEM) or N (%). Comparisons by univariate binary logistic regression or Fisher Exact test. *HCP contacts: Health Care professional contact(s) in the previous 4 weeks, not including any contact leading to referral to tertiary care. #Origin of referral not available for 2 patients.

**Table 2.** Demographics and biochemistry data for patients referred from primary care or who self-presented to hospital, related to family history of T1DM

| | Primary Care | Primary Care | Self-Presented | Self-Presented |
|---|---|---|---|---|
| | No Family History | Family History | No Family History | Family History |
| N | 201 | 15 | 28 | 17 |
| Age (y) | 8.9 (0.3) | 9.9 (0.9) | 9.2 (0.8) | 9.8 (0.8) |
| Female | 98 (48.8%) | 9 (60%) | 12 (42.9%) | 4 (23.5%) |
| BMI | 17.2 (0.3) | 19.9 (1.1) | 16.9 (0.6) | 17.8 (0.7) |
| DKA at presentation | 52 (25.9%) | 1 (6.7%) | 17 (60.7%) | 1 (5.9%) |
| Moderate or Severe DKA | 25 (12.4%) | 1 (6.7%) | 13 (46.4%) | 1 (5.9%) |
| pH | 7.32 (0.01) | 7.36 (0.03) | 7.19 (0.04) | 7.35 (0.02) |
| Bicarbonate (mmol/L) | 20.4 (0.1) | 22.2 (0.7) | 12.9 (1.4) | 23.1 (1.2) |
| Glucose (mmol/L) | 26.8 (0.7) | 26.1 (2.8) | 31.0 (1.7) | 22.7 (2.0) |
| HbA1c (mmol/mol) | 110.9 (1.9) | 98.4 (9.1) | 107.6 (6.3) | 93.0 (7.0) |
| Ethnicity | | | | |

| | | | | |
|---|---|---|---|---|
| NZ European | 125 | 9 | 11 | 14 |
| Maori | 28 | 2 | 4 | 3 |
| Pacific Islander | 24 | 3 | 8 | 0 |
| Other | 24 | 1 | 5 | 0 |
| Deprivation Score | 4.6 (2.8) | 5.2 (3.7) | 6.1 (3.0) | 4.6 (3.2) |

Data are mean (SEM) or n (%). Origin of referral unknown for 2 patients.

Table 3. Factors associated with health care professional (HCP) contacts in the 4 weeks before presentation in new patients with T1DM

| | No recent HCP contact | Recent HCP contact | P-value |
|---|---|---|---|
| N | 230 | 33 | |
| Age (y) | 9.2 (0.3) | 8.0 (0.7) | 0.11 |
| BMI | 17.3 (0.3) | 17.8 (0.7) | 0.5 |
| % Female | 47% | 45% | 0.87 |
| Ethnicity | | | |
| NZ European | 146 (63.5%) | 14 (42.4%) | 0.023 |
| Maori | 31 (13.5%) | 7 (21.2%) | |
| Pacific Islander | 28 (12.1%) | 7 (21.2%) | |
| Other | 25 (10.9%) | 5 (15.2) | |
| Family history of T1DM | 32 (13.9%) | 1 (3%) | 0.11 |
| Primary care referral | 192 (83.5%) | 24 (72.7%) | 0.11 |
| Diagnostic testing before referral | 70 (30.4%) | 16 (48.5%) | 0.08 |
| Deprivation Score | 4.6 (0.2) | 5.7 (0.5) | 0.06 |
| DKA | 23.5% | 51.5% | 0.001 |
| Moderate or Severe | 29 (12.6%) | 11 (33%) | 0.003 |

| | | | |
|---|---|---|---|
| DKA | | | |
| pH | 7.33 (0.01) | 7.23 (0.03) | <0.001 |
| Bicarbonate (mmol/L) | 20.3 (0.4) | 16.6 (1.5) | 0.004 |
| Glucose (mmol/L) | 26.6 (0.6) | 29.5 (1.7) | 0.1 |
| HbA1c (mmol/mol) | 110.2 (1.9) | 98.4 (4.9) | 0.035 |

Comparison by binary logistic regression. Data are mean (SEM) or n (%)