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#### **Brief report**

# Use of a standardised diabetic ketoacidosis management protocol improved clinical outcomes

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#### ABSTRACT

We analysed the clinical outcomes of using a standardised protocol in the management of diabetic ketoacidosis. Of 71 admissions, the protocol group (n = 35) had significantly shorter length of hospitalisation, shorter time to normalise bicarbonate, fewer incidence of hypokalaemia and hypoglycaemia compared with the control group (n = 36).

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#### 1. Introduction

Diabetic ketoacidosis (DKA) is a common, acute metabolic complication of diabetes mellitus and can lead to significant morbidity and mortality if not effectively managed. Mortality in the last decade is reported to be 0.2–2% in developed countries [1–3]. Management of DKA includes rehydration, insulin, replacement of electrolytes especially potassium, correction of acidosis and treatment of the precipitating

factor [1,4]. Several health professionals are usually involved in the management of DKA especially after hours using variable regimens. Over the last decade, efforts to standardise DKA management have been shown in other countries to have a positive impact on patient care [5–7] but it has not been reported in an Australian setting.

We aimed to analyse whether using a standardised protocol improves clinical outcomes in the acute management of DKA at a tertiary teaching hospital in Australia.

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#### 2. Patients and methods

Admissions with DKA in patients aged ≥16 year for the period January 2008–March 2012 were identified from the Townsville Hospital medical records. 40 consecutive admissions managed according to the protocol (January 2010–March 2012) and another 40 admissions managed by non-standardised practice between January 2008 and December 2009 who served as control were reviewed using medical notes and electronic pathology results. Patients who self-discharged against medical advice before resolution of acidosis and those who stayed in hospital for more than 96 h without ongoing issues with DKA (5 in the protocol group and 4 in the control group) were excluded.

The protocol was adapted from the Scottish National DKA protocol [8] and modified to meet local practice need. It involved fluid resuscitation with 0.9% sodium chloride and an initial fixed rate (6 units/h) of intravenous insulin. When blood glucose level (BGL) became <14 mmol/L, 10% dextrose (100 ml/h) was introduced and the IV insulin rate was reduced to 3 units/h to maintain BGL 9–14 mmol/L. Potassium replacement was commenced when serum potassium was <5 mmol/L. Subcutaneous insulin was given at least 2 h before cessation of IV insulin when serum bicarbonate was  $\geq$ 20 mmol/L and patient tolerated oral food. Patients could be discharged if stable for at least 4 h on subcutaneous insulin.

The main parameters that were compared between the protocol and control groups included mean time taken to normalise serum bicarbonate (time between the start of IV insulin and the time at which the serum bicarbonate was first

measured as ≥20 mmol/L), mean length of hospitalisation (recorded time at emergency triage to the time of discharge), incidence of hypoglycaemia, incidence of hypokalaemia, mean total IV insulin dose, mean duration of IV insulin, average BGL reduction in first 3 h, and mean amount of IV fluid.

Data were compared using two-tailed Student's t-test, Chisquare and Mann–Whitney U tests as applicable. Quantitative data were expressed as mean  $\pm$  SD.

#### 3. Results

A total of 35 and 36 admissions were included in the protocol and control groups respectively for analysis. The baseline characteristics between the two groups were comparable (Table 1). Compared with the control group, the protocol group had significantly shorter mean time taken to normalise serum bicarbonate (15.1 h in protocol vs. 24.6 h in control) (P = 0.01), and mean length of hospitalisation (37.9 h vs. 49.2 h) (P = 0.01). Subgroup analysis of mean time taken to normalise serum bicarbonate in patients with initial bicarbonate ≤10 mmol/L showed that the protocol group took 11.4 h less than the control group (P = 0.05). Incidence of hypokalaemia and hypoglycaemia were significantly lower in the protocol group; 28.6% in the protocol group vs. 52.8% in the control group for hypokalaemia (P = 0.038), and 8.6% in the protocol group vs. 28% in the control group for hypoglycaemia (P = 0.036). There was no significant difference in the mean total IV insulin dose, total amount of IV fluid or average BGL reduction (Table 2).

Characteristics	Protocol $(n = 35)$	Control $(n = 36)$	P value
Mean age (yrs)	$30\pm13$	27 ± 8	P = 0.19
Gender	n (%)	n (%)	P = 0.73
Male	15 (43%)	14 (39%)	
Female	20 (57%)	22 (61%)	
Aetiology	n (%)	n (%)	P = 0.30
Missed insulin	14 (40%)	17 (47%)	
Infection	10 (29%)	13 (36%)	
New diagnosis of DM	2 (6%)	3 (8.5%)	
Alcohol	6 (17%)	3 (8.5%)	
Other	3 (8%)	- '	
Type of DM	n (%)	n (%)	P = 0.1
Type 1	34 (97.1%)	34 (94.5%)	
Type 2	1 (2.9%)	2 (5.5%)	
Mean blood glucose on presentation	$28.68\pm11.93\ \text{mmol/L}$	$28.61 \pm 9.6~\mathrm{mmol/L}$	P = 0.98
Ketones on presentation	n (%)	n (%)	P = 0.16
Small	4 (2%)	1 (3%)	
Moderate	2 (6%)	1 (3%)	
Large	29 (82%)	31(86%)	
No record	<u> -                                   </u>	3 (8%)	
Serum bicarbonate (mmol/L) on presentation	n (%)	n (%)	P = 0.64
10 or less	16 (46%)	18 (50%)	
10.1–15	10 (29%)	12 (33%)	
More than 15	9 (25%)	6 (17%)	
Mean level	$11.6 \pm 6.06$ mmol/L	$10.5 \pm 5.25$ mmol/L	P = 0.42

Blocked insulin pump in 1, unknown precipitant in 2 admissions.

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