



## Original Research

## Examining the “Killer K” of Diabetic Ketoacidosis at a Tertiary Care Hospital: An Exploratory Study

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## ABSTRACT

**Objectives:** Hypokalemia, a frequently cited complication of diabetic ketoacidosis (DKA) treatment, can have critical implications, including arrhythmias and death. We assessed the prevalence of hypokalemia and its associated factors in patients with DKA at our tertiary-care centre and identified opportunities to improve care.

**Methods:** We conducted a retrospective chart review to establish the prevalence of hypokalemia in patients diagnosed with DKA between July 2012 and July 2013. A focused root-cause analysis was subsequently performed to identify Canadian Diabetes Association DKA clinical practice guideline deviations and preventable errors that resulted in significant hypokalemia ( $K < 3.3$  mmol/L) during the first 48 hours of management. Clinical and management details were reviewed to determine the type, preventability and root cause(s) of each error.

**Results:** We identified 40 cases of DKA during the study period. The overall prevalence of hypokalemia during DKA treatment was 38% (15/40), with 25% in type 1 and 56% in type 2 diabetes. Males were more likely to experience hypokalemia (87%), and 47% of hypokalemic incidents occurred in the first presentation of diabetes. All 10 cases of significant hypokalemia were reviewed. We identified 23 errors in 6 (60%) cases, of which 87% were deemed to be preventable. The most common errors were noncessation of insulin infusion during hypokalemia (60%), inadequate potassium supplementation (50%) and infrequent biochemical monitoring (50%).

**Conclusions:** Hypokalemia occurs frequently during acute DKA management and is often preventable. Our findings suggest that interventions targeted at enhancing awareness of guidelines may reduce hypokalemia rates.

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## R É S U M É

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sécurité du patient  
amélioration de la qualité

**Objectifs :** L'hypokaliémie, une complication qui est fréquemment rapportée lors du traitement de l'acidocétose diabétique (ACD), peut avoir des conséquences cruciales, dont les arythmies et la mort. Nous avons évalué la prévalence de l'hypokaliémie et ses facteurs associés chez les patients souffrant d'ACD de notre centre de soins tertiaires et défini les possibilités d'amélioration des soins.

**Méthodes :** Nous avons mené une revue rétrospective de dossiers pour établir la prévalence de l'hypokaliémie chez les patients ayant reçu un diagnostic d'ACD entre juillet 2012 et juillet 2013. Nous avons subséquemment réalisé une analyse centrée sur les causes fondamentales pour déterminer les écarts et les erreurs évitables des lignes directrices de pratique clinique sur l'ACD de l'Association canadienne du diabète qui entraînaient une hypokaliémie importante ( $K < 3,3$  mmol/l) durant les 48 premières heures de la prise en charge. Nous avons passé en revue les données cliniques et de prise en charge pour déterminer le type, l'aspect évitable, et la ou les causes fondamentales de chacune des erreurs.

**Résultats :** Nous avons relevé 40 cas d'ACD durant la période de l'étude. La prévalence globale de l'hypokaliémie au cours du traitement de l'ACD était de 38% (15/40), soit 25% du diabète de type 1 et

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56% du diabète de type 2. Les hommes étaient plus susceptibles d'avoir une hypokaliémie (87%), puis 47% des épisodes hypokaliémiques sont apparus au premier tableau clinique du diabète. Nous avons passé en revue les 10 cas d'hypokaliémie importante. Nous avons trouvé 23 erreurs dans 6 (60%) cas et avons considéré que 87% de ces dernières étaient évitables. Les erreurs les plus fréquentes étaient le maintien de la perfusion d'insuline au cours de l'hypokaliémie (60%), l'apport supplémentaire inadéquat en potassium (50%) et la surveillance biochimique peu fréquente (50%).

**Conclusions :** L'hypokaliémie qui apparaît fréquemment au cours de la prise en charge de l'ACD en phase aiguë est souvent évitable. Nos résultats suggèrent que les interventions visant à améliorer la connaissance des lignes directrices peuvent réduire les taux d'hypokaliémie.

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## Introduction

Diabetic ketoacidosis (DKA) is a potentially life-threatening hyperglycemic emergency that leads to severe metabolic derangements, including ketone production, metabolic acidosis and electrolyte disturbances (1). Hypokalemia, in particular, is a feared complication of DKA treatment.

Causes of hypokalemia are multifold but can include hyperglycemia-driven osmotic diuresis resulting in urinary potassium loss and gastrointestinal losses in some patients. Insulinopenia impairs potassium entry into cells and results in many patients' with DKA presenting with initially high or normal serum potassium levels despite having whole-body potassium deficits. The use of intravenous fluids and insulin as part of DKA management promotes an additional loss of potassium in urine and the shift of potassium into cells, leading to a drop in serum potassium concentrations. Without early adequate potassium supplementation, hypokalemia may arise and can have critical implications, including respiratory muscle weakness, cardiac arrhythmias and even death (1–3).

Although hypokalemia in the context of DKA treatment has been reported to range from 24% to 81% (4–11), no study has examined the demographics, clinical characteristics or predisposing and precipitating causes of hypokalemia in this specialized population. In this study, we sought to assess the prevalence and factors linked to hypokalemia in patients with DKA at our tertiary-care centre, with the goal of defining opportunities for systematic changes to improve care.

## Methods

### Study design

We conducted a retrospective chart audit of all patients treated for DKA from July 1, 2012, to July 1, 2013, at St. Michael's Hospital, an inner-city academic centre with an 80-bed general internal medicine service and a 24-bed medical/surgical intensive care unit. Charts were reviewed to establish the prevalence of hypokalemia ( $K < 3.5$  mmol/L) in the first 48 hours following presentation to the emergency room (ER). A focused root-cause analysis was subsequently performed to identify preventable errors that resulted in significant hypokalemia ( $K < 3.3$  mmol/L) during the first 48 hours of DKA management. This study was approved by the Research Ethics Board at St. Michael's Hospital.

### Identification of cases and definition of hypokalemia

All patients over the age of 18 admitted to St. Michael's Hospital (under any admitting service) with the most responsible admission diagnosis of DKA (as defined by International Classification of Diseases [ICD]-9 classification [250.1] and by ICD-10 classification [i.e. E10.1]) from July 1, 2012, to July 1, 2013) were included in the study sample. Cases with hypokalemia were identified from the hospital's electronic medical record database.

A hypokalemic incident meriting a preliminary investigation was defined to have occurred if serum potassium concentration was  $< 3.5$  mmol/L. Significant hypokalemia was defined as a serum potassium concentration of  $< 3.3$  mmol/L, with concentrations  $< 2.5$  mmol/L classified as severe hypokalemia, based on expert consensus.

### Data collection

Hospital records were reviewed to determine the demographic, clinical and treatment characteristics of all patients diagnosed with DKA during the study period. Variables included patient demographics (age, gender, type of diabetes); serum potassium values; serum glucose values; hospital length of stay; and in-hospital mortality. To minimize the effect of poor documentation, multiple health record sources were utilized, including ER physician and nursing notes, consultation notes, daily progress notes, ward-based nursing notes, discharge summaries and electronic medical record-based investigations. At our institution, ER physicians typically initiate DKA management, but the majority of the subsequent acute care is referred to either the internal medicine or intensive care medicine resident. Endocrinology consult teams are typically not involved in the initial acute care but are consulted to assist with transition to subcutaneous insulin and to provide diabetes-related education.

The primary outcome assessed was the prevalence of overall hypokalemia. Cases with any hypokalemic incident were further evaluated to ascertain details concerning the use and timing of intravenous insulin, the amount of potassium supplementation, urine output and biochemical monitoring, the time to closure of anion gap (AG) and the normalization of acidosis as well as incidence of DKA recurrence during the same admission. Counterbalance outcome measures (measures related to or affected by changes in practice or improvement cycle) were also collected, including incidences of hyperkalemia (serum potassium concentration  $> 5$  mmol/L) and severe hyperkalemia (serum potassium concentration  $> 6$  mmol/L). Hyperkalemic values were derived only from nonhemolyzed samples. The incidence of concurrent hypokalemia and hyperkalemia, defined as an individual's experiencing both hypokalemic and hyperkalemic incidents during the first 48 hours following presentation to the ER for DKA, was also collected.

For each index patient who experienced significant hypokalemia, a detailed chart review was conducted to identify diabetes-related characteristics (e.g. glycated hemoglobin [A1C] levels, diabetes duration, vascular complications) as well as epidemiologic risk factors for DKA and hypokalemia. Vascular complications were categorized as either microvascular (e.g. neuropathy, nephropathy or retinopathy) or macrovascular (e.g. cerebrovascular disease, coronary artery disease or peripheral vascular disease). Assessed risk factors for DKA included new diagnosis of diabetes mellitus, insulin omission, insulin infusion pumps, infection, myocardial infarction, abdominal crisis, trauma, thyrotoxicosis, atypical antipsychotics or interferon use, and history of cocaine use. Conditions that relayed an increased risk for hypokalemia were also examined and included starvation, hypothermia, vomiting, diarrhea, offending medications (e.g. loop/thiazide diuretics, beta agonists), salt-wasting

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