

Updates in the Management of Diabetic Ketoacidosis

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ABSTRACT

Diabetic ketoacidosis (DKA) is an emergency for people with diabetes characterized by hyperglycemia, metabolic acidosis, and ketosis. DKA onset and recurrence can largely be prevented through patient education. Nurse practitioners are well positioned to promote patient education, self-management, and individualized patient care. This article outlines updates in the clinical management of patients with DKA to optimize care and reduce costs.

Keywords: diabetes, diabetes mellitus, diabetic ketoacidosis, endocrine emergency, hyperglycemia

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Diabetic ketoacidosis (DKA) is a metabolic derangement characterized by hyperglycemia, metabolic acidosis, and ketosis.¹ DKA occurs in patients with diabetes who have a lack of circulating insulin relative to physiologic requirement, such as in type 2 diabetes mellitus (T2DM), or absolute depletion, such as in type 1 diabetes mellitus (T1DM),² in the presence of increased counterregulatory hormones (cortisol, growth hormone, epinephrine, and glucagon). The lack of adequate insulin can occur from medication noncompliance, infection, or a precipitating pathologic event, such as a myocardial infarction, that causes an increased metabolic demand for insulin.

The Centers for Disease Control and Prevention (CDC) United States Diabetes Surveillance System recently reported an increase in DKA episodes in the US between 2009 and 2014, with an average annual increase of 6.3%.³ DKA can occur at any age but primarily occurs in those aged younger than 30 years (36% incidence) and between 30 and 50 years (27% incidence).² The highest incidence of DKA is for those aged between 11 and 15 years.⁴ In addition, girls and women and the immigrant population are at higher risk.

In the past, DKA had a fatality rate of 1% to 5%. Older adults and individuals who have comorbid risk factors are in the highest risk category.¹ In recent years, however, the overall mortality of DKA has

declined due to earlier detection and increased evidence-based management. In fact, the recent CDC US Diabetes Surveillance System report indicated that the mortality for DKA has decreased to 0.4%.³

Episodes of DKA typically require an emergency department visit or hospital admission for the patient to receive insulin, intravenous (IV) fluids, and electrolyte correction. Hospital encounters can be costly, with recent reports suggesting that a single hospital encounter for DKA treatment can cost up to \$17,500,⁵ with an average length of stay of 3.4 days. Further, the annual direct and indirect cost of DKA treatment in the US exceeds \$2.3 billion.¹

DKA and recurrent DKA are largely preventable through better outpatient management, patient education, and promoting self-care behaviors.⁶ Nurse practitioners (NPs) have an opportunity to educate patients about the risks of DKA, promote self-management, and offer patient-centered care. Presented here is a review the management of DKA and updates in clinical care.

DIAGNOSIS

The diagnosis of DKA is made based on the metabolic triad of high blood glucose (BG) levels (generally > 250 mg/dL), acidosis (pH < 7.2), and the presence of urine or serum ketones (Table).⁵ Inpatient providers commonly rely on laboratory data

Table. Diagnosis of Diabetic Ketoacidosis⁷

Variable	Diabetic Ketoacidosis		
	Mild	Moderate	Severe
Arterial pH	7.25-7.3	7- < 7.24	< 7.00
Serum bicarbonate, mEq/L	15-18	10- <15	<10
Urine ketones	Positive (trace or higher)	Positive	Positive
Serum ketones	Positive	Positive	Positive
Anion gap, mEq/L	>10	>12	>12
Mental status	Alert	Alert/drowsy	Stupor/coma

to confirm DKA, whereas outpatient providers rely on history, presentation, BG levels, and urine ketones.

Common presenting symptoms include abdominal pain and the classic triad of hyperglycemia symptoms: polydipsia, polyphagia, and polyuria. Physical examination findings can include any or all of tachycardia, hypotension, Kussmaul respirations, significant dehydration, or a change in mental status.⁷

It is important to consider differential diagnoses of metabolic acidosis that may include lactic acidosis or hyperchloremic acidosis. Differential diagnoses for ketosis include starvation ketosis (dietary history, weight trends) or alcoholic ketoacidosis (alcohol consumption history), hyperemesis, isopropyl alcohol, or ketotic hypoglycemia.¹

Euglycemic DKA (euDKA), which occurs when the patient presents with acidosis and ketosis but has a glucose ≤ 200 mg/dL, has become an emerging concern. Causes of euDKA can include recent insulin administration, decreased caloric intake, substantial alcohol consumption, chronic liver disease, or rarely, glycogen storage issues.⁸ In addition, there have been increasing reports of euDKA caused by a new class of drugs for diabetes, sodium glucose cotransporter 2 (SGLT-2) inhibitors. In May 2015, the US Food and Drug Administration added a warning about the risk of DKA with use of these drugs. One study suggested that the risk of DKA for patients using SGLT-2 inhibitors was twice as high as those prescribed a dipeptidyl peptidase IV inhibitor, after controlling for other risk factors, although the risk of hospitalization was low.⁹ The exact cause of this relationship is unknown, but several theories include reduced insulin doses when SGLT-2 is

initiated, an increase in glucagon, or decreased excretion of ketone bodies.¹ Other related factors may be mild infection, increased activity, reduced food intake, or insulin reduction or omission.¹⁰ Case reports of traditional and euDKA occurring while using SGLT-2 inhibitors has been shown in patients with T1DM and in those with T2DM.¹¹ Many patients with euDKA present with nausea and vomiting but are misdiagnosed due to the lack of clear glucose elevation.

DIAGNOSTIC WORKUP

Providers in outpatient settings where laboratory results are not readily available should rely on BG and ketone values for the initial diagnosis. BG values will typically be > 250 mg/dL, although up to 10% of patients in DKA may present with euDKA.⁹ For this reason, some experts argue that the cutoff BG value for diagnosing DKA should be decreased to 200 mg/dL.¹²

Ketone measurement is an important diagnostic component and severity classification of DKA for patients in the outpatient setting. Urine ketones will test positive, although may be as minimal as “trace” ketones. Urine dipsticks measure acetoacetate (AcAc) and acetone but not β -hydroxybutyrate (β -OHB). Given this, the measurement of AcAc in the urine tends to underestimate the severity of DKA, because the ratio of AcAc to β -OHB can be 1:10 during ketoacidosis.¹³ Other limitations with urine ketone testing include lag time in change in urine ketones, difficulty obtaining urine from dehydrated patients, and subjective measurements by the patient. Many experts agree that measurement of blood or capillary ketones is preferred due to these limitations.⁹

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