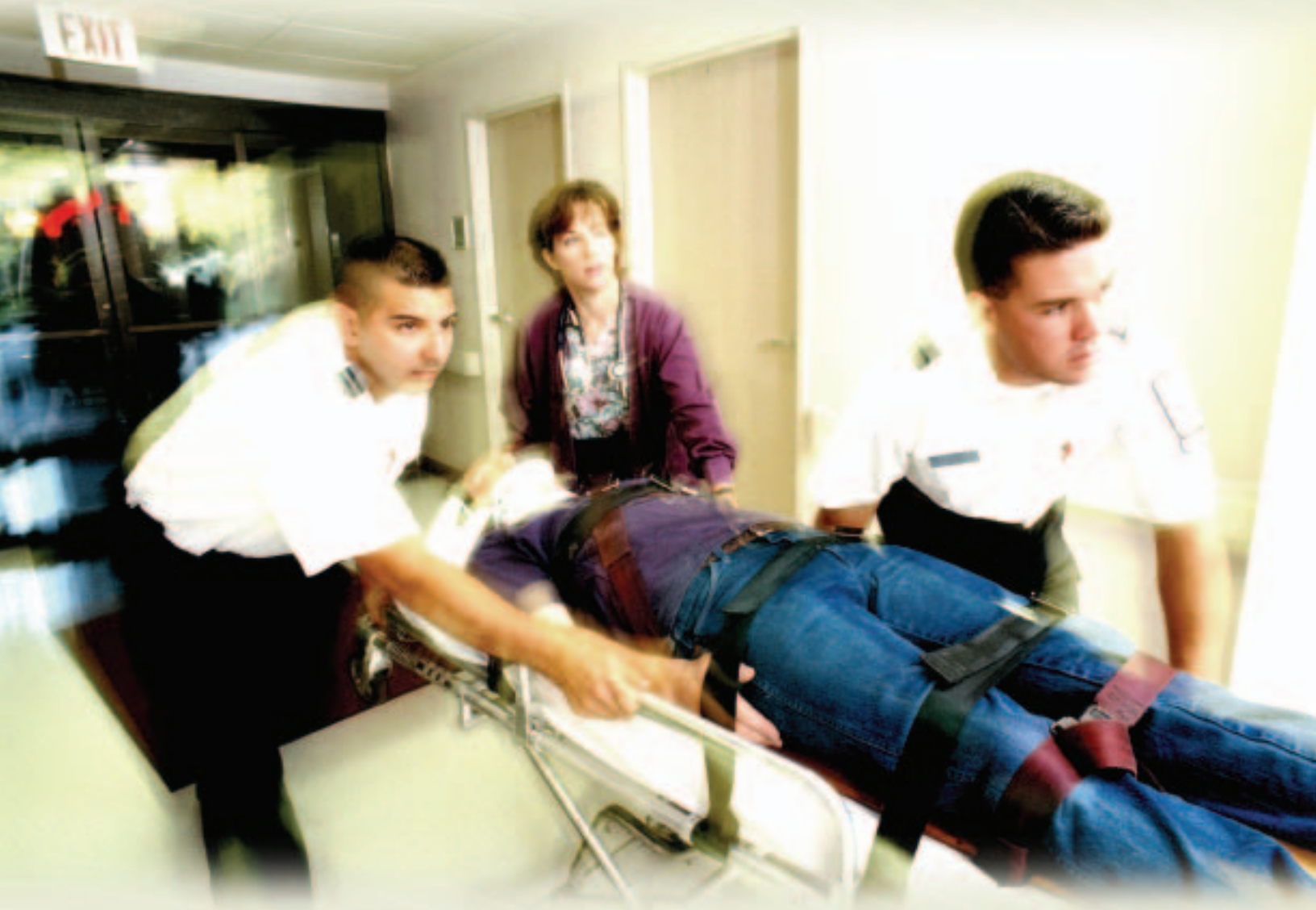
A Continuing Medical Education Monograph for Physicians

# A Comprehensive Review of Diabetic Ketoacidosis and Hyperosmolar Hyperglycemic State in Adults

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PIM Postgraduate Institute for Medicine

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## PROGRAM GOAL

This monograph is a review of the pathophysiology, diagnosis, treatment, and complications of diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) in adults.

## TARGET AUDIENCE

This educational activity has been designed to meet the educational needs of physicians involved in the management of patients with diabetes.

## PURPOSE

Provide physicians treating patients with diabetes the latest information about the pathophysiology, diagnosis, and management of DKA and HHS to help reduce the incidence, morbidity, and mortality of the disorder.

## STATEMENT OF NEED/ PROGRAM OVERVIEW

DKA and HHS are life-threatening emergencies and are the most serious complications of diabetes. DKA is caused by relative or absolute insulin deficiency, which results in hyperglycemia, ketonemia, dehydration, electrolyte imbalances, and acidosis. Treatment protocols for adults generally advocate a more rapid and aggressive reversal of DKA than is advised for children. DKA consumes a significant proportion of all direct medical costs for adults and children with type 1 diabetes. The management of DKA requires a full complement of hospital, emergency, and intensive care services. In the United States, more than 100,000 individuals are hospitalized each year for DKA and the mortality rate is 2% to 5%.

Most cases of DKA can be prevented by using an effective diabetes management plan that includes patient self-care. Early identification of precipitating signs and symptoms and prompt, appropriate intervention can reduce the frequency of DKA episodes that result in a medical emergency.

The healthcare team must stay informed about the recent developments in diabetes care and have the necessary clinical skills to prevent and manage DKA episodes. Through good self-management and intensive glycemic control, the incidence, morbidity, and mortality of DKA can be reduced.

## EDUCATIONAL OBJECTIVES

After completing this educational activity, participants should be able to:

- Differentiate between DKA and HHS
- Describe the epidemiology and pathophysiology of DKA
- Identify the signs, symptoms, and complications of DKA
- Discuss methods of treatment and recovery care for DKA
- Describe potential complications during treatment of DKA and special considerations for pregnant and elderly individuals
- Provide strategies for the prevention of DKA

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EXIT

# INTRODUCTION

Each year more than 100,000 people are hospitalized for diabetic ketoacidosis (DKA) in the United States, resulting in cumulative annual hospital costs that may be more than $1 billion.[1] DKA is a life-threatening illness, and the mortality rate for DKA, estimated between 2% to 5%, has remained relatively unchanged since the 1970s.[2,3] The average length of hospital stay for patients diagnosed with DKA, however, has declined from 7.9 days in 1980 to 3.9 days in 2002.[4] More than 25% of the costs for direct medical care for adults with type 1 diabetes covers DKA; this increases to 50% in those experiencing recurrent episodes.[5]

Among the benefits of effective glycemic control is reduced incidence of DKA or hyperosmolar hyperglycemic state (HHS) and associated morbidity and mortality. Both DKA and HHS are life-threatening emergencies.[6,7] The American Diabetes Association (ADA) recommends that adults with diabetes maintain preprandial plasma glucose levels between 90 and 130 mg/dL, postprandial levels <180 mg/dL, and an A1C <7% while pointing out that more stringent glycemic goals (ie, a normal A1C <6%) may further reduce complications at the cost of increased risk of hypoglycemia.[8] The American College of Endocrinology has somewhat different targets, recommending preprandial plasma glucose levels ≤110 mg/dL, postprandial levels ≤140 mg/dL, and an A1C ≤6.5%.[9]

Comprehensive education in self-management is a critical element for achieving and maintaining optimal glycemic control. The physician and patient must set realistic treatment goals with the diabetes management team and family members. An effective program requires ongoing support from the clinical care team.

Healthcare professionals who treat patients with diabetes must know how to prevent and manage DKA. This monograph defines DKA and describes symptoms and warning signs, diagnosis, current practices, complications, prevention methods, special circumstances, and economic issues.

# DEFINITION

DKA is a reversible but life-threatening complication that results from relative or absolute insulin deficiency.[10] In the absence of insulin, most cells cannot use glucose. Fat breakdown provides an alternative source of energy. These metabolic abnormalities will, if not reversed, culminate in DKA, which is characterized by the presence of hyperglycemia, ketosis, and acidosis (Figure 1). The common signs and symptoms of DKA include "fruity" (acetone) breath secondary to ketosis, nausea, vomiting, polydipsia, and polyuria. Breathing becomes deep and rapid as the body attempts to correct the acidosis (Kussmaul's respiration). HHS is also a life-threatening condition arising from insufficiency of insulin action, leading to increased levels of counterregulatory hormones, alterations in osmolality, and hyperglycemia; however, HHS lacks the marked lipolysis, ketonemia, and acidosis associated with DKA.[1] DKA and HHS represent two extremes on the spectrum of decompensated diabetes; they differ by magnitude of hyperglycemia, severity of acidosis/ketonemia, and degree of dehydration.[7] Table 1 summarizes the diagnostic criteria distinguishing DKA from HHS.

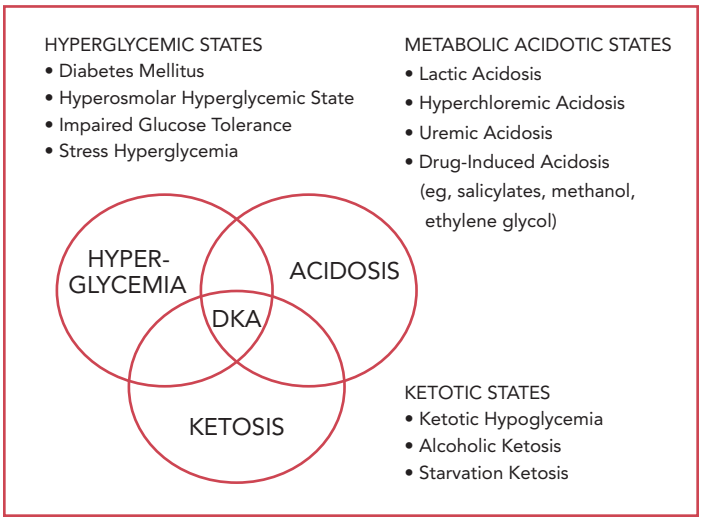


**Figure 1. Diagnostic triad of diabetic ketoacidosis (DKA).**
From Kitabchi AE. Diabetes mellitus. In: Glew RH, Peters SP, eds. *Clinical Studies in Medical Biochemistry*. New York, NY: Oxford University Press; 1987:102–117.

Both DKA and HHS can occur in patients with type 1 or type 2 diabetes.[1] DKA frequently affects young patients with type 1 diabetes on initial presentation; however, it does occur in patients with type 2 diabetes and in adults as well.[1,3,11–14] HHS occurs most commonly in older adults with type 2 diabetes.[13,15] In HHS, insulin resistance and relative insulin deficiency, which are usually a result of a precipitating factor (eg, stroke, infection, myocardial infarction), can result in high levels of blood glucose. Because HHS generally occurs in patients with type 2 diabetes who are capable of some insulin production, marked ketosis and attendant acidosis are not usually presenting clinical features. HHS and DKA can coexist, however, which can make differential diagnosis difficult.[16] HHS is often triggered by glucosuric diuresis.[17] The onset of DKA is typically more acute than that of HHS, with

symptoms often developing over a course of hours, as opposed to days to weeks for HHS.[1,13,18] The severity of DKA depends on the magnitude of the decrease in arterial pH, serum bicarbonate, and mental state rather than hyperglycemia (Table 1).

The severe hyperglycemic and hyperosmolar (concentrated serum) state associated with HHS typically leads to profound dehydration and confusion or coma. The excessive water loss through osmotic diuresis can be a vicious cycle, which can only be compensated by adequate fluid replacement.[16] Common signs of HHS include stupor, dehydration, and hypotension. The neurologic signs can range from aphasia to seizures or coma. Patients often present with weakness, visual disturbance, or leg cramps.[17]

**Table 1. Diagnostic Criteria for Diabetic Ketoacidosis (DKA) and Hyperosmolar Hyperglycemic State (HHS).**

| | DKA | | | HHS |
|---|---|---|---|---|
| | Mild | Moderate | Severe | |
| Plasma glucose (mg/dL) | >250 | >250 | >250 | >600 |
| Arterial pH | 7.25–7.30 | 7.00–7.24 | <7.00 | >7.30 |
| Serum bicarbonate (mEq/L) | 15–18 | 10 to <15 | <10 | >15 |
| Urine ketones* | Positive | Positive | Positive | Small |
| Serum ketones* | Positive | Positive | Positive | Small |
| Effective serum osmolality (mOsm/kg)† | Variable | Variable | Variable | >320 |
| Anion gap‡ | >10 | >12 | >12 | Variable |
| Alteration in sensoria or mental obtundation | Alert | Alert/ Drowsy | Stupor/ Coma | Variable Stupor/Coma |

*Nitroprusside reaction method; †calculation: 2(measured Na [mEq/L]) + glucose (mg/dL)/18; ‡calculation: $(Na^+) - (Cl^- + HCO_3^-)$ (mEq/L).



Most patients with severe DKA have some alteration in their level of consciousness.

## EPIDEMIOLOGY

A diagnosis of diabetes is often made following a severe DKA or HHS episode.[17,19,20] DKA episodes can often be attributed to the lack of early recognition and timely treatment of diabetes.[21,22] Approximately 20% to 30% of DKA cases[19] and 30% to 40% of HHS cases[7] occur in patients with newly diagnosed diabetes. Although DKA occurs most often in type 1 diabetes, patients with type 2 diabetes may develop DKA after many weeks of symptomatic hyperglycemia. In a retrospective medical record review, 39% of patients who presented with DKA had type 2 diabetes.[23] There were 4.6 to 8 cases of DKA per 1000 person-years compared with 1 case of HHS.[7]

Infections and inadequate insulin therapy (resulting from both inadvertent errors and intentional nonadherence with therapy) are major precipitating causes of DKA,[24–26] as are certain drugs, alcohol abuse, pancreatitis, cerebrovascular accident, myocardial infarction, stress, and trauma.[1,3,10,12,21,24,25,27] Psychosocial issues, such as the fear of hypoglycemia and weight gain, are sometimes involved in cases of undertreatment or outright omission of insulin.[1] In another retrospective medical record review, 55% of patients who presented with DKA had type 2 diabetes with infection being the most common precipitating cause of DKA.[28] In contrast, poor compliance with insulin therapy was the most frequent cause of DKA in patients with type 1 diabetes.[28] Many patients with type 2 diabetes who develop DKA have no obvious precipitating causes other than prolonged omission of oral hypoglycemic therapy.[23]

## PATHOPHYSIOLOGY

Insulin deficiency triggers a complex metabolic process. This process may begin insidiously and develop during a period of hours to days. Type 1 diabetes is characterized by a severe insulin deficiency, which results from autoimmune destruction of pancreatic β-cells. Type 1 diabetes is usually diagnosed in individuals younger than 30 years of age, although up to 10% of people with new-onset diabetes in adulthood at any age have type 1 diabetes. Insulin deficiency and hyperglycemia are usually not present until ~90% of insulin secretory capacity is lost.[10]

DKA occurs because of relative or absolute insulin deficiency coupled with a concomitant elevation of counterregulatory hormones. Fleckman's seesaw analogy describes the hormonal interplay that characterizes DKA (Figure 2).[29] Insulin, on one side of the seesaw, normally counterbalances the effect of the counterregulatory hormones (glucagon, catecholamines, cortisol, and growth hormone) on the opposite side.

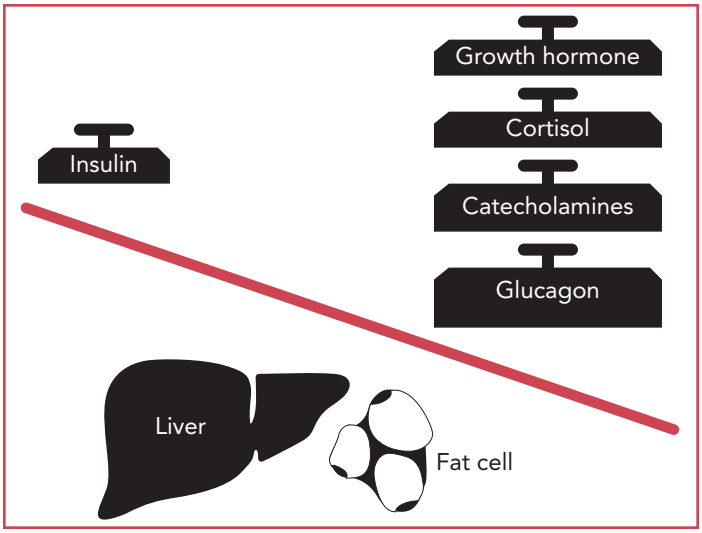


**Figure 2. Fleckman's seesaw analogy for diabetic ketoacidosis. Insulin is outweighed by the counterregulatory hormones.**

From Fleckman AM. Diabetic ketoacidosis. *Endocr Metab Clin North Am.* 1993;22:181–207.

Table 2 summarizes the effects of insulin and the counterregulatory hormones.[30] A relative or absolute deficiency of insulin and an excess of counterregulatory hormones result in: (1) gluconeogenesis, (2) glycogenolysis, (3) inhibition of peripheral glucose utilization, (4) lipolysis, and (5) stimulation of ketogenesis.

## Events

**Hyperglycemia.** Hyperglycemia occurs secondary to insulin deficiency, gluconeogenesis, and underutilization of glucose by the peripheral tissue. Insulin deficiency stimulates glucagon release, which contributes to the development of DKA by stimulating gluconeogenesis and ketogenesis. In times of stress, epinephrine stimulates glucagon release. The presence of increased concentrations of epinephrine, cortisol, and growth hormone accentuates the impairment of peripheral glucose utilization.[30]

> Glucagon plays an important role in the development of DKA because it influences gluconeogenesis and ketogenesis.

**Dehydration.** Patients with severe DKA may have fluid deficits of 6 liters or more with an average weight loss of 5% to 10% of their total body weight.[31] This may develop over a 12- to 24-hour period.[10] Dehydration occurs secondary to osmotic diuresis, which results from glucosuria.

Glucosuria develops when the blood glucose concentration exceeds the renal threshold (160–180 mg/dL) for glucose. The loss of water and dehydration are secondary to polyuria. Hyperventilation and vomiting may also contribute to fluid loss. The degree of dehydration can be assessed by determining the patient's weight loss. Otherwise, dehydration must be assessed by clinical signs such as tachycardia, orthostatic hypotension, and dry mucous membranes.

**Ketone production.** Ketogenesis occurs in a state of insulin deficiency and counterregulatory hormone excess, which results in ketonemia and ketonuria. Insulin deficiency leads to the breakdown of fat in adipose tissue (lipolysis) with the release of free fatty acids. Ketones are formed from free fatty acids in the liver. The pathophysiologic basis of DKA is shown in Figure 3. Insulin is required for the suppression of ketone production and the correction of acidosis. Insulin inhibits glycogenolysis and gluconeogenesis, suppresses lipolysis, and facilitates the conservation of sodium and other electrolytes by the kidney.[10]

**Table 2. Effects of Insulin and Counterregulatory Hormones.**

| | Gluconeogenesis *Liver* | Ketogenesis *Liver* | Glucose Utilization *Muscle* | Lipolysis *Adipose Tissue* |
|---|---|---|---|---|
| Insulin | ↓ | ↓ | ↑ | ↓ |
| Glucagon | ↑ | ↑ | → | → |
| Epinephrine | ↑ | ↑ | ↓ | ↑ |
| Cortisol | ↑ | ↑ | ↓ | ↑ |
| Growth hormone | → | ↑ | ↓ | ↑ |

↑Increased; ↓Decreased; →Minimal or no effect.

From Ennis ED, Stahl E, Kreisberg RA. Diabetic ketoacidosis. In: Porte D Jr, Sherwin RS, eds. *Ellenberg & Rifkin's Diabetes Mellitus.* 5th ed. Stamford, Conn: Appleton & Lange; 1997:829.

**Ionic changes.** Ketoacids are excreted as sodium and potassium salts, which contribute to the electrolyte disturbances seen in DKA. In an attempt to correct or buffer the acidosis, potassium ions move from the intracellular space to the extracellular space as hydrogen ions move to the intracellular space. As a result of the

potassium shift to extracellular fluid and urinary potassium loss, patients with DKA have a deficit of total body potassium. However, initial laboratory values may show low, normal, or high values of potassium.

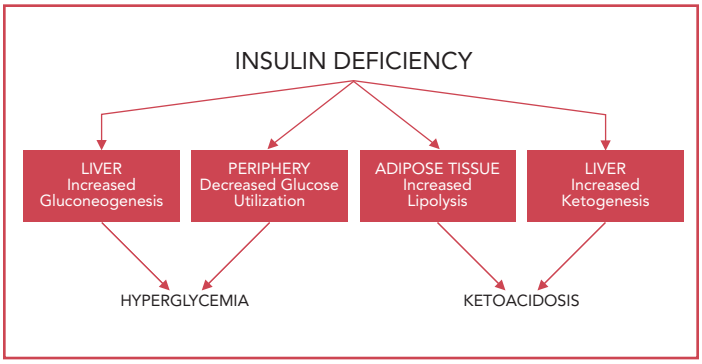


**Figure 3. Pathophysiologic changes due to insulin deficiency.**

## Compensatory Attempts

Excessive ketone production causes metabolic acidosis. DKA occurs when the increased production of ketoacids overwhelms attempts at compensation. The rapid overproduction of ketoacids depletes the buffering effect of bicarbonate and results in acidosis. The body uses three defense mechanisms in an attempt to counter impending acidosis: (1) respiratory compensation, (2) intracellular buffering, and (3) renal correction.[19,32] Excess carbon dioxide is exhaled to correct the metabolic acidosis. This results in tachypnea or Kussmaul's respiration. In addition, buffering occurs when excess hydrogen ions move intracellularly in exchange for potassium ions to maintain a neutral intracellular charge. The kidneys attempt to correct ketoacidosis by increasing the excretion of ketoacids. Acidosis will continue to worsen if not corrected by the administration of insulin.

Ketogenesis may rapidly overwhelm all attempts at compensation.

# TRIGGERS OF DKA AND HHS

DKA can be precipitated by many conditions that result in insufficient circulating levels of insulin or lead to the development of insulin resistance (Figure 4). Some of these conditions may promote transient hyperglycemia in patients without established diabetes. DKA may occur in patients with new-onset diabetes or in previously diagnosed patients who do not exercise proper self-care (eg, omission of or inadequate doses of insulin) or are not being properly managed.

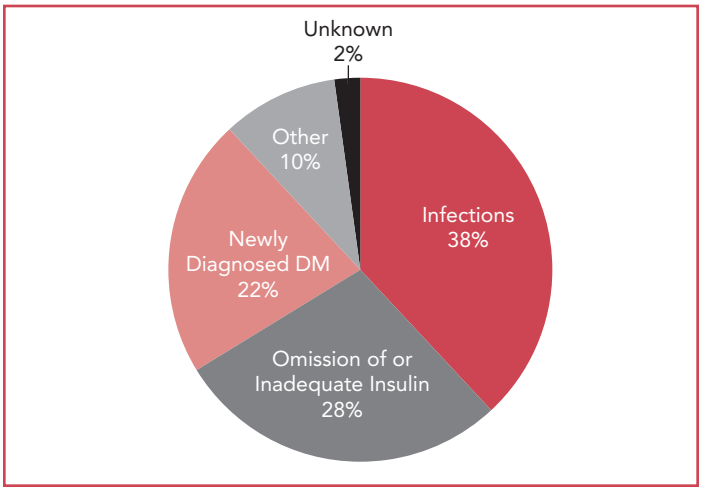


**Figure 4. Precipitating diabetic ketoacidosis factors in 202 patients admitted to the University of Tennessee – Memphis, Clinical Research Center.** DM = diabetes mellitus. From Kitabchi AE, Fisher JN, Murphy MB, Rumbak MJ. Diabetic ketoacidosis and hyperglycemic, hyperosmolar nonketotic state. In: Kahn CR, Weir GC, eds. *Joslin's Diabetes Mellitus.* 13th ed. Baltimore, Md: Lippincott Williams & Wilkins, 1994:738–770.

Intercurrent medical illness is the triggering event in 50% to 60% of adult cases of DKA.[30] Unfortunately, intercurrent illness is often erroneously accompanied by the discontinuation of insulin.[30] A precipitating factor may not be identified in ~20% to 30% of DKA cases.[21] To prevent DKA, it is important for the healthcare team to understand factors that may precipitate an event. These factors include the following:

- Infection is the most common precipitating factor in the development of DKA and HHS.[1,17] It stimulates the release of counterregulatory hormones, which promote gluconeogenesis and glycogenolysis. Cytokines (eg, interleukin-1) are increased and may also be implicated.[32]
- Errors in administration of insulin or oral antidiabetic (OAD) agents as well as drugs that reduce insulin secretion or increase insulin resistance may trigger DKA and HHS.[17,26] These include deliberate or inadvertent omission of insulin or OAD agents and inappropriate dosing of insulin during sick days. DKA may also result from inappropriately withholding or reducing the dose of insulin in a patient who is vomiting and unable to eat.
- Drugs that affect carbohydrate metabolism. β-Blockers, thiazides, and loop diuretics can reduce insulin secretion whereas corticosteroids can increase insulin resistance.[15]

- Use of atypical antipsychotic agents (eg, clozapine, olanzapine, quetiapine, or risperidone) may significantly impair glucose metabolism and lead to DKA.[33]
- Cardiovascular events can occur as a complication of DKA or may trigger DKA because of acutely increased insulin requirements. Cardiovascular events are a major cause of DKA-associated death. Myocardial infarction should always be considered a possibility in an elderly patient with DKA.[34] In some patients, warning signs or symptoms of acute myocardial infarction may be absent. This could result in a treatment delay because the patient may not seek medical attention at the critical time.
- Substance abuse causes about 10% of the DKA cases. Patients with diabetes who are under the influence of alcohol or illicit drugs may not be able to administer insulin appropriately, which can result in insulin deficiency and DKA.[35]
- DKA secondary to an increase in counterregulatory hormones can develop during pregnancy. The second and third trimesters of pregnancy are associated with increased insulin requirements and insulin resistance.[36]
- Psychiatric problems or any dramatic emotional responses to stress can contribute to the development of DKA secondary to increased levels of counterregulatory hormones.
- Fasting and dehydration can contribute to DKA.
- Insulin infusion pump malfunction can lead to an interruption in insulin delivery and result in DKA.[37] In the Diabetes Control and Complications Trial, DKA events were higher in the insulin pump group (1.8 events/100 patient-years) when compared with the multiple-dose insulin group (0.8 event/100 patient-years). Complete insulin deficiency occurs in patients with type 1 diabetes within a few hours after a pump malfunction. Pump patients must understand the importance of maintaining appropriate insulin delivery to minimize the risk of DKA. Good self-care can prevent pump malfunction in the majority of cases. Routine monitoring of blood glucose and attentiveness to symptoms of hyperglycemia should help pump users promptly identify insulin infusion problems (eg, empty insulin reservoir, displaced needle, improper placement of the pump reservoir, and infusion line blockage). Inflammation or infection at the injection site can be minimized by adhering to good hygiene and appropriate changes of the catheter insertion site.
- Other precipitating factors of DKA include pancreatitis, cerebrovascular accident, trauma, and drugs that affect carbohydrate metabolism (thiazide diuretics, corticosteroids).[1]

## WARNING SIGNS AND SYMPTOMS

Clinical signs and symptoms of DKA may include polyuria, polydipsia, vomiting, "fruity" (acetone) breath, dehydration, abdominal pain, and hyperventilation (Table 3). Symptoms of DKA may mimic other disease states or medical conditions. Nonspecific symptoms include lethargy, malaise, headache, and weakness. DKA should be considered in any ill person with diabetes, especially if nausea and vomiting are present. Although the clinical diagnosis of DKA is usually clear in a patient with type 1 diabetes, DKA should not be ruled out in an ill, elderly patient with type 2 diabetes.

**Table 3. Common Symptoms and Signs of Diabetic Ketoacidosis.**

| **Symptoms** | |
|---|---|
| • Nausea and vomiting | • Abdominal pain |
| • Thirst and polyuria | • Visual disturbances |
| • Weakness and/or anorexia | • Somnolence |
| **Signs** | |
| • Tachycardia | • Weight loss |
| • Hypotension | • Warm, dry skin |
| • Hyperpnea or Kussmaul's respiration | • Impaired consciousness and/or coma |
| • "Fruity" breath (odor of ketones) | • Dehydration |



Vomiting may signal an advanced stage of DKA. Polydipsia cannot compensate for dehydration. Urinary output continues because of persistent osmotic diuresis. Alterations in mental status, ranging from drowsiness to coma, may be seen in 10% to 20% of all patients with DKA.[38] Electrolyte and metabolic abnormalities seen in DKA include:

- pH <7.3

- Hypokalemia (usually after therapy has been initiated)
- Hypernatremia or hyponatremia
- Hyperosmolality
- Hyperglycemia
- Hypertriglyceridemia

## DIAGNOSIS

The diagnosis of DKA must be made rapidly to prevent morbidity and mortality. It can be difficult to make a diagnosis in the elderly. The patient may present with some or all of the symptoms previously discussed. The diagnosis can be made after obtaining a medical history and physical examination. The patient's medical history should be taken with special attention paid to recent infections, compliance with insulin or OAD therapy, concomitant medication use, and concurrent medical illnesses. It is important for the physician to obtain thorough information about the patient's diabetic history. If the patient is comatose, family members should be carefully questioned.

Hypotension, tachycardia, and tachypnea may be present. A rectal temperature measure may be required if tachypnea precludes oral measurement. An elevated body temperature warrants a careful examination and evaluation for possible infection. However, patients may not be febrile, even in the presence of an underlying infection.[38] A subnormal temperature can result from vasodilation. Hypothermia is an alarming sign and may be associated with increased risk of death.[38] Dehydration may result in poor skin turgor, warm dry skin, dry mucous membranes, tachycardia, and orthostatic hypotension. A change in systolic pressure of more than 10 mm Hg represents a fluid volume deficit and may be indicative of systemic dehydration. Urinary output may decrease to less than 30 mL/hr and the patient may develop anuria.[38]

DKA must be differentiated from other causes of acidosis. The diagnosis of DKA can be confused with alcoholic ketoacidosis or starvation ketosis because both conditions cause ketonemia and acidosis. Isolated alcoholic ketoacidosis is usually characterized by mild to moderate metabolic acidosis, an increased anion gap, and normoglycemia or hypoglycemia. Starvation ketosis is usually characterized by a normal arterial pH, a mildly increased anion gap, and the absence of significant ketonemia.[16] The diagnostic criteria and typical total body deficits in DKA are listed in Table 4.

**Table 4. Diagnostic Criteria and Typical Total Body Deficits in Diabetic Ketoacidosis.**

| | |
|---|---|
| **Diagnostic criteria*** | |
| Blood glucose | >250 mg/dL (13.9 mmol/L) |
| Arterial pH | <7.3 |
| Serum bicarbonate | <15 mEq/L |
| Urinary ketone† | Positive |
| Serum ketone | Positive at 1:2 dilutions |
| Serum osmolality | Variable |
| | |
| **Typical deficits** | |
| Water | 6 L or 100 mL/kbw |
| Sodium | 7–10 mEq/kbw |
| Potassium | 3–5 mEq/kbw |
| Phosphate | ~1 mmol/kbw |

kbw = kilogram body weight.
*Not all patients will meet all diagnostic criteria, depending on hydration status, previous administration of diabetes treatment, and other factors.
†Nitroprusside reaction method.
From Kitabchi AE, Wall BM. Management of diabetic ketoacidosis. *Am Fam Physician.* 1999;60:456.

### Anion Gap

Arterial pH is a measure of the acidity or alkalinity of the blood. The arterial pH in a patient with DKA reflects the degree of respiratory compensation and severity of acid-base disturbance. In DKA, ketoacids ionize at physiologic pH and the hydrogen ion of the ketoacids is buffered essentially mole for mole by bicarbonate, which results in the consumption and decrease in the serum bicarbonate.

The anion gap is the difference between serum cations and anions. In the circulation, $Na^+$ is the predominant cation while $Cl^-$ and $HCO_3^-$ are the main anions. During ketogenesis, metabolic acidosis occurs because bicarbonate concentration is reduced, resulting in an increased anion gap. In DKA, the increase in the anion gap is usually equivalent to the decrease in the bicarbonate concentration.

The anion gap is calculated by subtracting the sum of the chloride and bicarbonate concentrations from the "uncorrected" serum sodium concentration:

$[Na^+ – (Cl^- + HCO_3^-)]$

**Normal Individual**

Na, 140; Cl, 105; $HCO_3$, 27

Anion gap: (140 – [105 + 27]) = 8

An anion gap of 8 is normal.

**Patient with DKA**

Na, 130; Cl, 98; $HCO_3$, 10

Anion gap: (130 – [98 + 10]) = 22

This anion gap of 22 is elevated.

The range for normal anion gap can vary depending upon the laboratory performing the test. Normal values are guidelines and specific to each laboratory.

Table 5 lists average laboratory findings in DKA and compares them with average normal laboratory values.[30]

## TREATMENT GUIDELINES

The goal of treatment is to reverse the underlying metabolic abnormalities, restore intravascular volume, improve tissue perfusion, and normalize the serum glucose. Emergency treatment of DKA and HHS involves correcting dehydration, hyperglycemia, and electrolyte imbalances. One of the mainstays of treatment for DKA and HHS is fluid replacement.[17,18] The degree of hyperglycemia, acidosis, dehydration, and impairment of consciousness may vary depending on the severity of metabolic derangement, nutritional status, duration of DKA, concomitant medications and illnesses, and the degree of insulin deficiency. The causes should also be identified and treated with steps to prevent recurrence.[18]

The reversal of these abnormalities should be undertaken with meticulous care and frequent monitoring to correct and avoid serious electrolyte imbalances and fluid overload. Appendix A provides an example of a DKA flow sheet used by healthcare professionals to record treatment and successive changes in the patient's status. During the first 12 hours of therapy, the frequency of reevaluation depends on the patient's condition. Typically, the reevaluation is hourly for the first 4 hours and continues every 2 to 4 hours, depending on the patient's condition.

The healthcare team may follow a "critical pathway," a comprehensive guide for treatment that includes anticipated interventions and expected outcomes. Appendix B is an example of a critical pathway used to guide a multidisciplinary team through the management of DKA. These protocols improve the quality of care received by patients and are cost-effective.[38] The ADA guidelines for fluid replacement, insulin administration, and potassium replacement in adult patients with DKA are depicted in Figure 5. Adequate rehydration restores and maintains intravascular volume and preserves adequate renal blood flow. Insulin enhances the intracellular movement of potassium during the treatment of DKA.

**Table 5. Average Laboratory Findings in Diabetic Ketoacidosis (DKA) Versus Average Normal Laboratory Values.**

| Parameter | DKA* | Normal Values† |
|---|---|---|
| Plasma glucose (mg/dL) | 475 | 60–110 |
| Serum osmolality (mOsm/kg) | 309 | 275–293 |
| Sodium (mEq/L) | 131 | 135–145 |
| Potassium (mEq/L) | 4.8 | 3.5–5.0 |
| $HCO_3^-$ (mEq/L) | 9 | 23–29 |
| BUN (mg/dL) | 21 | 8–20 |
| Arterial pH | <7.3 | 7.35–7.45 |
| Ketonuria | ≥3+ | NA |
| Growth hormone (ng/mL) | 7.9 | 0–5 |
| Cortisol, 8 a.m. (µg/dL) | 49 | 5–20 |
| Free fatty acids (mmol/L) | 2.26 | 0.19–0.9 |
| Glucagon (pg/mL) | 400–500 | 50–200 |
| Lactate, plasma (mmol/L) | 4.6 | |
| Venous | | 0.5–2.02 |
| Arterial | | 0.5–1.6 |
| β-Hydroxybutyrate (mmol/L) | 13.7 | NA |
| Catecholamines, total free (µg/mL) | 1.78 ± 4 | 4–126 µg/24 hr |

BUN = blood urea nitrogen.

*All average laboratory values for DKA were taken from Ennis ED, Stahl E, Kreisberg RA. Diabetic ketoacidosis. In: Porte D Jr, Sherwin RS, eds. *Ellenberg & Rifkin's Diabetes Mellitus.* 5th ed. Stamford, Conn: Appleton & Lange; 1997:831.

†All normal laboratory values were taken from Spraycar M, ed. *Stedman's Medical Dictionary.* 26th ed. Philadelphia, Pa: Williams & Wilkins; 1995:1992–2010; Beers MH, Berkow R, eds. *The Merck Manual of Diagnosis and Therapy.* 17th ed. Centennial Edition. 1999. Available at: http://www.merck.com/pubs/mmanual/. Accessed May 6, 2002; and Tierney LM Jr, McPhee SJ, Papadakis MA, eds. *Current Medical Diagnosis & Treatment.* 41st ed. New York, NY: The McGraw-Hill Companies; 2002:1711–1719.

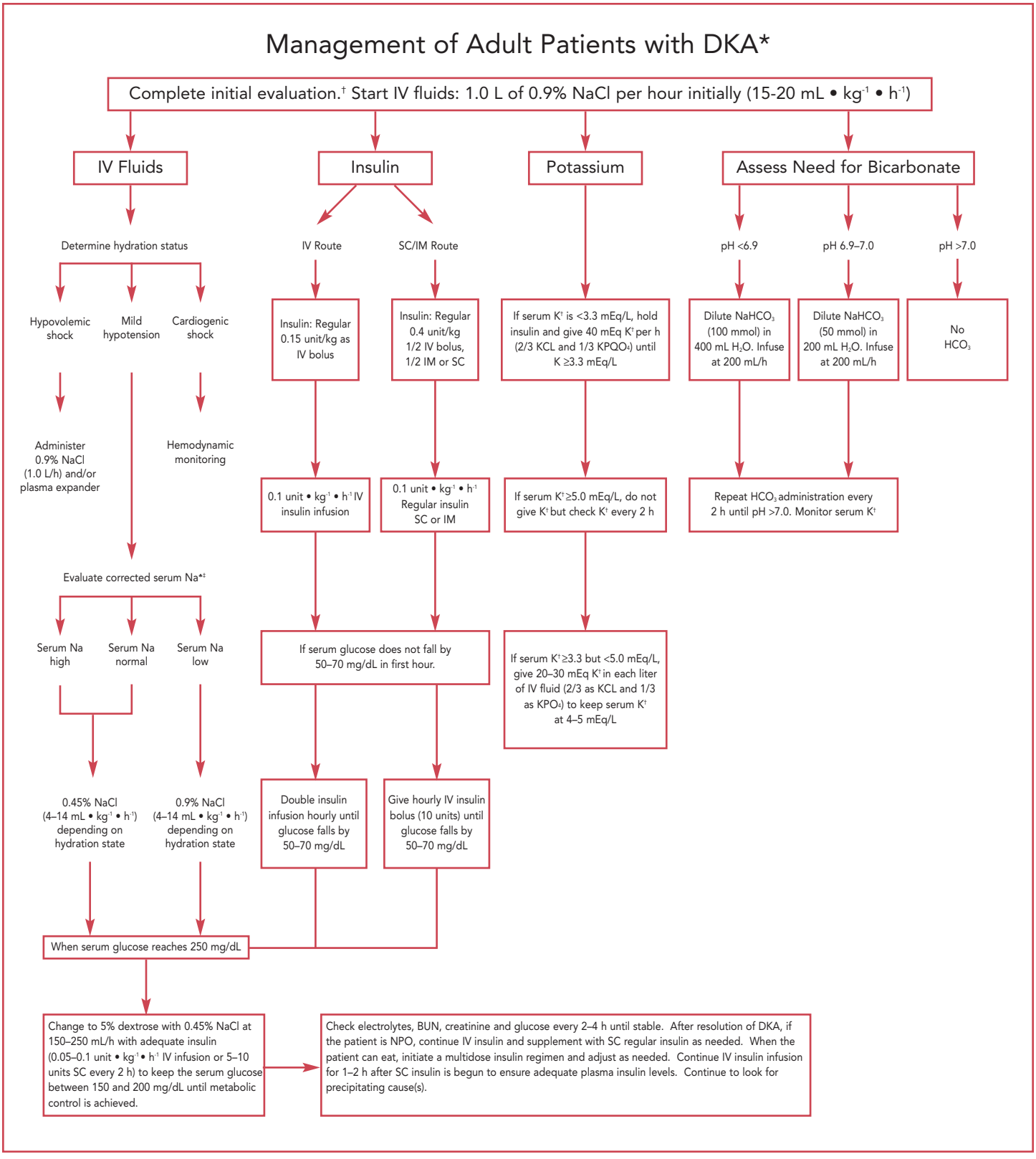


**Figure 5. A flow diagram for the management of adult patients with diabetic ketoacidosis.**

IM = intramuscular; IV = intravenous; SC = subcutaneous

*Diagnostic criteria for DKA include: blood glucose >250 mg/dL, arterial pH <7.3, bicarbonate <15 mEq/L, with moderate ketonuria or ketonemia.

†After patient history and physical examination, obtain arterial blood gases, urinalysis, complete blood count with differential, blood glucose, blood urea nitrogen (BUN), chemistry profile, electrolytes, and creatinine levels, as well as an electrocardiogram. Chest x-ray and cultures should be obtained as needed.

‡Serum sodium should be corrected for hyperglycemia (for each 100 mg/dL glucose >100 mg/dL, add 1.6 mEq to sodium value for corrected serum sodium value).

The ADA guidelines for insulin therapy in DKA are depicted in Table 5. Because of its quick onset of action, regular insulin by continuous intravenous infusion is the preferred treatment for DKA in adults unless the episode is mild. Following exclusion of hypokalemia, the ADA guidelines recommend administration of an intravenous bolus of regular insulin at 0.15 unit/kg body weight, followed by continuous infusion of regular insulin at a dose of 0.1 unit/kg per hour. This will usually decrease plasma glucose concentration by 50–75 mg/dL per hour, which is similar to a higher dose insulin regimen (Figure 5). If the plasma glucose levels do not decrease by at least 50 mg/dL from the original value in the first hour, the hydration status should be checked. If the hydration status is acceptable, then the insulin infusion may be doubled every hour until plasma glucose shows a steady decline of 50 to 75 mg/dL per hour. Once the plasma glucose reaches 250 mg/dL, the insulin infusion rate can sometimes be decreased to 0.05 to 0.1 unit/kg per hour, and 5% to 10% dextrose can be added to the intravenous fluids.[1]

Modern management of DKA emphasizes the use of lower doses of insulin to avoid hypoglycemia and other complications of treatment.

Timely potassium replacement and careful monitoring during therapy for DKA are essential to avoid potentially life-threatening hypokalemia. Extra caution is necessary in patients with severe hypokalemia associated with dehydration and hyperglycemia to avoid potentially fatal cardiac arrhythmias. Potassium is generally initiated after serum levels decrease <5.0 mEq/L (see Figure 5). Potassium replacement is started earlier in patients with serum potassium levels <3.3 mEq/L, exhibiting electrocardiogram (ECG) signs of flat or inverted T waves, depressed ST segments, and emergence of U waves. Both insulin and bicarbonate therapy lower serum potassium levels, and in cases where hypokalemia is significant, or when serum potassium levels are <3.3 mEq/L, insulin therapy should be postponed until serum potassium has been adequately replenished.[1]

The risks and benefits of sodium bicarbonate replacement are listed in Table 6. Bicarbonate levels increase during insulin therapy. The use of bicarbonate in DKA is controversial. Bicarbonate is generally not recommended for the treatment of DKA when pH is ≥7.1.[10] If pH is <7.0, bicarbonate may be administered (see Figure 5). Some physicians recommend administering bicarbonate when patients are severely acidotic, especially if hypotension, shock, or arrhythmias are present.[10] However, no prospective, randomized studies have shown benefits associated with bicarbonate administration.[1]

**Table 6. Benefits and Risks of Sodium Bicarbonate Replacement.**

| |
|---|
| **Benefits** |
| • May reduce extracellular acidosis |
| **Risks** |
| • Accelerated reduction in plasma potassium concentration |
| • Exacerbation of intracellular acidosis |



Patients with DKA are usually phosphate-depleted, secondary to decreased food intake, excessive catabolism, and increased urinary excretion. However, serum phosphate levels are often normal or increased at the time of presentation.[1] The administration of insulin enhances the intracellular movement of phosphate, which reduces the plasma phosphate concentration. Potassium phosphate can be administered cautiously during treatment to prevent hypophosphatemia, although there are no controlled trials demonstrating a definite benefit. Patients with anemia, cardiac problems, depressed respiration, or serum phosphate level <1.0 mg/dL, may be candidates for careful phosphate replacement.[1] Overadministration of phosphate can result in hypocalcemia; therefore, calcium levels should be checked both before phosphate is administered and during phosphate replacement.[10]

Aggressive fluid replacement is the most important step in treating HHS; patients can lose up to 25% body weight and require 8 to 12 liters over 48 hours.[15,17] An ADA protocol has been established for treating HHS.[1] There is general agreement that initial replacement should be with isotonic saline (0.9% at a rate of 1 L/hr for approximately 2 hours).[1,7] The goal of replacement is to replenish intravascular volume and correct hyperosmolality. Subsequent fluid and other requirements are based on hydration status, vital signs, serum electrolytes and glucose,

and urine output.[1,7] Potassium, sodium, and phosphate are replaced on the basis of laboratory measures. In some cases, anticoagulants may be needed to prevent venous thromboembolism. Insulin is administered after an adequate amount of fluid replacement.[15,17] Administering insulin before fluids can exacerbate hypotension and cause vascular collapse or death through insulin-induced cellular uptake of water.[17] Care in the selection of the intravenous fluid is critical as well. Whereas initial hydration in the setting of hypotension and tachycardia should be with blood, colloid, or isotonic fluids, as hemodynamic stability is achieved, most patients can be switched to hypotonic fluids such as one-half normal saline to avoid the hyperchloremic metabolic acidosis which often accompanies overhydration with normal saline. Comprehensive guidelines have been formulated by the ADA to treat both DKA and HHS.[24]

## RECOVERY CARE

Patients with DKA require intensive medical care, monitoring, follow-up, and education. Two important management principles are followed during the recovery period: (1) continue administering insulin; and (2) allow patients to eat. Intravenous fluids and short-acting insulin are continued until acidosis is corrected and the patient can ingest food without vomiting. Early feeding is considered because the added carbohydrate in the presence of insulin assists in the clearance of ketones. After the patient begins to eat, several days will still be required to correct all biochemical abnormalities associated with an episode of DKA.[10] The patient need not remain hospitalized beyond the time required to replete fluids and electrolytes, to ascertain and treat precipitating causes, and to demonstrate that the patient and caregivers can self-manage the diabetes.

When the decision is made to begin feeding, the patient is switched from intravenous to subcutaneous insulin. Since subcutaneous insulin acts more slowly than intravenous insulin, the transition to subcutaneous insulin is initiated with caution to avoid recurrence of acidosis during the transition phase. The insulin drip is discontinued 1 to 2 hours after the administration of subcutaneous insulin. Glucose levels are monitored 2 hours later and at least every 4 hours subsequently until a relatively stable subcutaneous insulin regimen is established. Therapy is usually required for 24 to 48 hours.[26] Resolution of DKA is indicated by blood glucose <200 mg/dL, a bicarbonate ≥18 mEq/L, and venous pH >7.3.[26]

## ELECTROLYTE MANAGEMENT DURING RECOVERY

Hyperchloremic acidosis with a normal anion gap is a common occurrence during recovery. During an episode of DKA, sodium is excreted as the sodium salt of ketoacids. Relative hyperchloremia can occur when treatment with intravenous solutions containing equal parts of sodium and chloride is used.[29] Because chloride losses are smaller than sodium losses, relative hyperchloremia occurs with therapy. The correction of DKA causes sodium bicarbonate to shift into the intracellular space leaving chloride overrepresented in the extracellular space.[29] If the anion gap gradually normalizes during therapy, a subsequent period of hyperchloremia-related nonanion gap acidosis is of no clinical concern.

## COMPLICATIONS

The majority of DKA cases are treated successfully without complications. However, potentially life-threatening complications are possible and include: hypoglycemia, hypokalemia, hyperchloremia, thromboembolic events, congestive heart failure, cerebral edema, and acute respiratory distress syndrome.

**Hypoglycemia** can occur if an excess of insulin is administered relative to glucose supply. Severe hypoglycemia can be a life-threatening complication. Sweating, tremors, and palpitations may occur with mild hypoglycemia; loss of consciousness and convulsions can occur with severe hypoglycemia. Patients should receive hourly monitoring of plasma glucose initially and after adjustments in intravenous insulin or administered carbohydrates to detect a rapidly decreasing blood glucose level and to prevent the development of hypoglycemia.

**Hypokalemia** is potentially life-threatening if potassium replacement is delayed or inadequate. Hypokalemia usually occurs after the initiation of insulin therapy, secondary to the intracellular movement of potassium. Increased potassium supplementation may be needed if bicarbonate therapy is required to correct ketoacidosis. Bicarbonate promotes the intracellular movement of potassium and increases the risk of hypokalemia.

**Hyperchloremia,** or hyperchloremic normal anion gap metabolic acidosis, is present in ~10% of patients admitted with DKA and is common in patients recovering from DKA.[24] It is usually caused by an excessive use of saline for fluid and electrolyte replacement during treatment.

However, other causes of hyperchloremia include loss of potential bicarbonate due to excretion of ketoanions as potassium and sodium salts, decrease in availability of bicarbonate in proximal tubule which leads to an increase in chloride reabsorption, and reduction of bicarbonate and other buffering capacities in other body compartments.[24]

**Thromboembolic events** may cause death in adults with DKA.[19] Prolonged stasis, immobility, and hemoconcentration are major precipitating factors of a thromboembolic event.[19] Antithrombotic therapy in the form of external compression devices or subcutaneous heparin is warranted in more severe or prolonged cases.

**Congestive heart failure** can develop with fluid replacement therapy, especially if an acute myocardial infarction or an underlying diabetic cardiomyopathy is missed.

**Cerebral edema** is a rare, often fatal complication that usually occurs within the first 4 to 24 hours after the initiation of therapy. It is more common in children, especially those younger than 4 to 5 years of age with DKA and new-onset diabetes. There are no established warning signs or clinical predictors.[38] Frequent ongoing neurologic assessments of the patient are critical during fluid replacement and insulin therapy.[38] Signs and symptoms may include headache, lethargy, abnormal pupil response, behavioral changes, seizures, bradycardia, papilledema, or unconsciousness.

> Cerebral edema is an extremely serious, although rare, complication of DKA and occurs more often in children than adults.

**Acute respiratory distress syndrome** is a form of acute lung injury that may occur as a result of various insults. This may lead to decreased intravascular osmotic pressure and fluid shifts resulting in pulmonary edema. Sepsis is the predominant risk factor.[39,40] Other predisposing conditions include multiple transfusions, severe nonthoracic trauma, pulmonary contusion, aspiration of gastric contents, multiple fractures, drug overdose, and pneumonia.[39,40] Caution in the rate of replacement of intravenous fluids should be exercised in patients with hypoxia and with suspected intrapulmonary or systemic infection.

## PREVENTION

Preventing DKA is one of the primary goals in diabetes management. The diabetes care team, including physicians, nurses, pharmacists, and diabetes educators, should train patients with diabetes to recognize the early signs and symptoms of DKA and to take immediate and appropriate action (see Table 3). When signs and symptoms suggestive of DKA occur, patients must contact a diabetes care team member promptly. Patients should perform and interpret self-monitoring blood glucose (SMBG) results and urine or blood ketone tests. Patients should minimize the risk of DKA by maintaining blood glucose levels as close as possible to their target range, following appropriate sick day management strategies (including notifying their diabetes care team in the event of a vomiting episode), and ensuring that insulin therapy is never omitted (Table 7).

### Table 7. Patient Self-Care for Sick Days.

- Prepare a personal sick day plan with your physician before you become ill
- When you are sick, always take your diabetes medicine
- Test your blood glucose level every 4 hours or more frequently if needed
- Test your urine or blood for ketones
- Call the doctor if
  - Your blood glucose is consistently >250 mg/dL
  - Your ketone test is moderate or high
  - You feel sick or vomit
  - You think you might have an infection
- Keep well hydrated
- Replace solid foods that contain starch and sugar (bread, fruit) with liquids that contain sugar (fruit juices, soft drinks) through the guidance of your diabetes care team

In a recent prospective study of adults with ketosis-prone diabetes, adherence with certain lifestyle modifications, including diet and exercise, and preserved β-cell function were found to predict good glycemic control (A1C <7.0%) one year subsequent to an initial episode of DKA.[41] The following types of intervention have reduced the number of episodes of DKA and the amount of healthcare resources used to treat DKA[5]:

- Patient education
- Comprehensive management programs
- Psychotherapy
- Treatment protocols
- Stepwise intervention programs
- Decentralized nurse-managed, pharmacist-managed, and physician-backed programs

It may be possible to identify risk factors by evaluating the patient's social environment. Patients with limited financial resources should be referred to a social worker for assistance. A referral to appropriate community resources and associations for support and education may help patients and their families.

The risk of DKA in patients who use continuous subcutaneous insulin infusion pumps can be reduced by limiting the use of these devices to patients who are highly motivated and properly trained in its usage. The patient must have a clear understanding of the pump function, exercise good hygiene, plan for sick days, adjust insulin appropriately, and recognize the signs and symptoms of DKA. Patients are instructed to perform frequent SMBG tests (at least 4 times a day), perform urine or blood ketone tests when appropriate, and respond appropriately to signs and symptoms of hyperglycemia and hypoglycemia. Healthcare professionals who are experienced in pump use should educate and guide these patients. Improved pump technology reduces the equipment malfunctions that can lead to DKA.

### Outpatient Prevention Measures

Intervention with supplemental injections of short-acting insulin or insulin analogs and oral administration of fluids are often initiated in patients with mild ketosis. In the absence of nausea and vomiting, patients with mild to moderate ketosis (glucose level >250 mg/dL, with moderate to large amounts of urinary ketones detected by urine ketone dipstick) may respond to supplemental doses of short-acting insulin or analogs, which can be repeated in 4 hours or less if the problem has not resolved. The development of nausea and vomiting or failure to clear ketones necessitates further evaluation in a hospital emergency room.

### Preventing Recurrent Diabetic Ketoacidosis

Frequent, recurrent DKA indicates the need for a detailed assessment by the diabetes care team. The team must determine whether the diabetes management plan is appropriate and identify any factors that promote noncompliance. Typical factors include:

- Inadequate patient education
- Cognitive impairment
- Substance abuse
- Lack of motivation
- Financial issues
- Emotional or psychiatric disorders
- Sensory impairment
- Problem with insulin/insulin delivery system

If patients or their families cannot achieve effective self-management, the team should focus on teaching them to identify the symptoms that require prompt medical attention. Patients who have repeated episodes of DKA may need psychological consultation.

## SPECIAL CIRCUMSTANCES

### Pregnancy

DKA occurs in 1% to 3% of pregnancies complicated by diabetes.[42] It is an acute medical emergency.[42] An episode of DKA lasting 2 to 5 hours or longer can result in the death of the fetus. DKA most commonly occurs in the second and third trimesters when increased insulin resistance is present. Prenatal and perinatal counseling is imperative in pregnant patients with diabetes. DKA frequently occurs when pregnancy appears in the presence of undiagnosed diabetes. Factors that predispose pregnant women to DKA are[43]:

- Accelerated starvation (eg, eating disorder)
- Dehydration secondary to vomiting
- Lowered buffering capacity
- Increased production of insulin antagonists
- Stress

Other precipitating factors of DKA were reviewed earlier (see "Triggers of DKA and HHS"). Fetal loss, the most serious consequence, occurs in about 9% of pregnant women presenting with DKA, even with the use of insulin and fluid therapy.[42] The mechanism of fetal mortality is unclear and could be related to fetal distress and hypoxia. Maternal ketoacids cross the placenta and cause fetal acidosis. Management of this condition is the same as for a nonparous individual.

### Elderly

There are special considerations involved in caring for the elderly with DKA because of the high mortality rate in this population. Elderly patients have an increased risk for fluid overload, decreased pulmonary capacity, silent cardiac events, and thromboembolic events.[38] It is critical that cardiopulmonary function be monitored, good pulmonary hygiene be practiced, and abdominal complaints be evaluated in the elderly. Initial therapy is usually conducted in an intensive care unit. Once patients are stabilized, they are transferred to a general unit.

## ECONOMIC ISSUES

The costs of DKA prevention with medication, monitoring SMBG, urine, and ketones, and patient education are a fraction of the cost of an emergency hospitalization. Location of treatment, intensive care facility versus general ward, plays a substantial role in the overall expense of DKA. The treatment of DKA requires a full complement of hospital, emergency, and intensive services, which consumes significant healthcare resources. This is especially true for patients hospitalized for multiple episodes. Treating a patient with DKA requires a full-time team of specialists to administer intravenous medications and fluids and perform careful hourly monitoring, frequent laboratory tests requiring rapid turnaround time and interpretation, and reliable bedside blood glucose monitoring.[44]

The newer rapid-acting insulin analogs, insulin aspart and insulin lispro, have been tested in therapy for DKA. Conclusions drawn from these studies have been that rapid-acting analogs, delivered subcutaneously every 1 to 2 hours, may provide an alternative to intravenous regular insulin for the treatment of mild and moderate DKA.[45,46] Although insulin analogs cost more than formulations of regular human insulin, the option of subcutaneous treatment could potentially reduce the cost of DKA therapy if it could be done in less intensive surroundings. However, the presumption that intravenous insulin therapy requires a critical care facility, whereas subcutaneous insulin injection therapy can be carried out in a less intensive setting, will not apply to all institutions.[44]

From population-based studies, the annual incidence rate for DKA is estimated to range from 4.6 to 8 episodes per 1000 patients with diabetes.[1] With the mean medical care for a patient with DKA estimated at $13,000 per episode, the overall hospital cost for patients with DKA may be more than $1 billion per year.[1,24] A substantial proportion of the cost for direct medical care for adults with type 1 diabetes is attributable to DKA. It is estimated that about one half of the DKA cost derives from patients who experience multiple episodes.[5]

Two studies show that specialists (endocrinologists) provide more cost-effective care compared with nonspecialists. Patients under the care of specialists have a shorter hospital stay, fewer medical procedures, lower medical costs ($10,109 for nonspecialist care vs $5463 for specialist care), and a lower rate of recurrent DKA.[47,48] Early conversion to oral feeding and subcutaneous insulin therapy are also associated with a shorter hospital stay.

## SUMMARY

DKA is a reversible but potentially life-threatening illness that results from relative or absolute insulin deficiency. Without insulin the body cannot utilize glucose as a fuel source and must obtain an alternative source of energy. This triggers a complex metabolic process that causes the breakdown of fat in adipose tissue and electrolyte disturbances that ultimately produces ketones and makes the blood acidic. The characteristics of DKA are hyperglycemia, ketosis, and acidosis. The clinical signs and symptoms include polyuria, polydipsia, progressive dehydration, and Kussmaul's respiration.

DKA usually can be prevented in the outpatient setting through effective patient education and timely intervention by the diabetes care team. Prompt and appropriate treatment of DKA most often results in satisfactory outcomes. Critical care personnel must know how to stabilize the patient's condition rapidly, administer appropriate medical treatment, and initiate a thorough evaluation to identify possible precipitating factors. Insulin administration, rehydration, and electrolyte replacement are the cornerstones of treatment. Early conversion to oral feeding and subcutaneous insulin therapy after the metabolic derangement is corrected is associated with a shorter length of hospital stay. Healthcare professionals who treat patients with diabetes should heed these important observations and recommendations:

- Treatment should be initiated immediately when DKA is diagnosed or suspected.
- An excellent clinical outcome is usually possible with meticulous care of patients with DKA.
- Successful treatment of patients with DKA is based on understanding that the etiology of DKA is severe insulin deficiency.
- Management of DKA includes administering insulin, correcting metabolic abnormalities by replacing fluids and electrolytes, identifying and treating precipitating causes, and monitoring for complications.
- Subcutaneous use of rapid-acting insulin analogs may provide an alternative to the use of intravenous insulin therapy in the treatment of mild and moderate DKA.
- Prevention and timely reversal of DKA can reduce morbidity and mortality and the cost of treatment.

# Appendix A

| Diabetic Ketoacidosis Flow Sheet | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Hour** | **0** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** | **10** | **11** | **12** |
| Weight (daily) | | | | | | | | | | | | | |
| Mental status* | | | | | | | | | | | | | |
| Temperature | | | | | | | | | | | | | |
| Pulse | | | | | | | | | | | | | |
| Respiration/Depth† | | | | | | | | | | | | | |
| Blood Pressure | | | | | | | | | | | | | |
| Serum Glucose (mg/dL)/L | | | | | | | | | | | | | |
| Serum Ketones | | | | | | | | | | | | | |
| Urine Ketones | | | | | | | | | | | | | |
| **Electrolytes** | | | | | | | | | | | | | |
| Serum Na (mEq/L) | | | | | | | | | | | | | |
| Serum K (mEq/L) | | | | | | | | | | | | | |
| Serum Cl (mEq/L) | | | | | | | | | | | | | |
| Serum $HCO_3$ (mEq/L) | | | | | | | | | | | | | |
| Serum BUN (mg/dL) | | | | | | | | | | | | | |
| Effective Osmolality‡ | | | | | | | | | | | | | |
| Anion Gap | | | | | | | | | | | | | |
| **Blood Acidity** | | | | | | | | | | | | | |
| pH Venous | | | | | | | | | | | | | |
| pH Arterial | | | | | | | | | | | | | |
| **Blood Gases** | | | | | | | | | | | | | |
| $PO_2$ | | | | | | | | | | | | | |
| $Pco_2$ | | | | | | | | | | | | | |
| $O_2$ Saturation | | | | | | | | | | | | | |
| **Insulin** | | | | | | | | | | | | | |
| Units (past hour) | | | | | | | | | | | | | |
| Route | | | | | | | | | | | | | |
| **Intake (past hour)** | | | | | | | | | | | | | |
| 0.45% Saline (mL) | | | | | | | | | | | | | |
| 0.9% Saline (mL) | | | | | | | | | | | | | |
| 5% Dextrose (mL) | | | | | | | | | | | | | |
| KCl (mEq) | | | | | | | | | | | | | |
| $PO_4$ (mg/dL) | | | | | | | | | | | | | |
| Other | | | | | | | | | | | | | |
| **Output** | | | | | | | | | | | | | |
| Urine (mL) | | | | | | | | | | | | | |
| Other | | | | | | | | | | | | | |

*A, Alert; D, Drowsy; S, Stupor; C, Comatose.
†D, Deep; S, Shallow; N, Normal.
‡2 x measured serum Na (mEq/L) + glucose (mg/dL)/18.

From Kitabchi AE, Fisher JN, Murphy MB, Rumbak MJ.
Diabetic ketoacidosis and the hyperglycemic, hyperosmolar nonketotic state. In: Kahn CR, Weir GC, eds. *Joslin's Diabetes Mellitus.* 13th ed. Baltimore, Md: Lippincott Williams & Wilkins; 1994:738–770.

# APPENDIX B

## CRITICAL PATHWAY

| ON ADMISSION | DAY 2 | DAY 3 | DAY 4 |
|---|---|---|---|
| **Health History**<br>• Medication history, immediate past illnesses or life events, time and amount of last insulin dose, nutritional patterns | | | |
| **Physical Assessment**<br>• Neuro checks to assess for cerebral edema<br>• Cardiovascular assessment: BP, HR, skin turgor<br>• Respiratory assessment: tachypnea, Kussmaul's respiration<br>• Musculoskeletal assessment: muscle weakness, fatigue<br>• GI assessment: nausea, vomiting, abdominal pain, bowel sounds<br>• Temperature | **Physical Assessment**<br>• Continue neuro checks to assess for cerebral edema<br>• Respiratory, cardiovascular, GI, and skin integrity assessments<br>• Vital signs every 2 hours<br>• Monitor for signs of hypoglycemia | **Physical Assessment**<br>• Respiratory, cardiovascular, GI, and skin integrity assessments<br>• Vital signs every 4 hours<br>• Monitor for signs of hypoglycemia | **Physical Assessment**<br>• Routine assessment and vital signs<br>• Monitor for signs of hypoglycemia |
| **Lab and Other Data**<br>• Initial serum glucose<br>• Bedside glucose monitoring every 1–2 hours; awareness of potential for hypoglycemia<br>• Serum ketones<br>• Urine ketones<br>• Potassium every 1–2 hours<br>• Phosphorus<br>• BUN/creatinine<br>• Bicarb<br>• ABGs<br>• CBC with differential<br>• Other electrolytes<br>• Blood cultures if indicated<br>• Intake and output | **Lab and Other Data**<br>• Continue bedside glucose monitoring every 2 hours<br>• Urine ketones<br>• Monitor electrolytes, especially potassium<br>• Intake and output | **Lab and Other Data**<br>• Continue bedside glucose monitoring every 2 hours<br>• Urine ketones<br>• Monitor electrolytes<br>• Intake and output | **Lab and Other Data**<br>• Continue bedside glucose monitoring q.i.d.<br>• Monitor electrolytes<br>• Intake and output |
| **Other Diagnostics**<br>• 12-lead ECG<br>• Continuous ECG monitoring | **Other Diagnostics**<br>• Continuous ECG monitoring if indicated; otherwise d/c | | |
| **Treatment**<br>• IV access<br>• NS @ 1 L/hr for 2–3 hours<br>• Replace potassium 10–30 mEq/hr<br>• Replace other electrolytes as indicated by lab data<br>• Regular insulin 0.1–0.2 U/kg per hour<br>• NG intubation<br>• Foley catheter<br>• Initiate appropriate antibiotic therapy as needed<br>• Antiembolic therapy | **Treatment**<br>• Change fluids to D5W or D51/2NS when serum glucose = 250–300 mg/dL<br>• Regular insulin 0.1 U/kg per hour<br>• Replace electrolytes as indicated by lab data<br>• NG tube to low intermittent suction<br>• Foley catheter<br>• Pulmonary hygiene<br>• Continue antiembolic therapy<br>• Begin education with family to prevent recurrence | **Treatment**<br>• Begin SC insulin injections 1–2 hours before discontinuing IV insulin. Include sliding scale<br>• Discontinue NG if bowel sounds present and patient is alert<br>• Begin oral feeding and advance diet as tolerated<br>• Dietary consultation, if indicated<br>• Discontinue the Foley catheter<br>• Up in chair and advance activity as tolerated<br>• Continue antiembolic therapy<br>• Review patient knowledge concerning sick day management, blood glucose and urine monitoring, signs and symptoms of hypoglycemia, and DKA<br>• Educate patient and family to prevent recurrence | **Treatment**<br>• Adjust SC insulin dosage as indicated as lab data. Include sliding scale<br>• ADA diet as prescribed<br>• Continue patient and family education to prevent recurrence<br>• Provide written instructions<br>• Consider home care referral as indicated |
| **Expected Outcomes**<br>• Hemodynamic stability<br>• Begin lowering of the blood glucose level<br>• Preventing complications of treatment and immobility | **Expected Outcomes**<br>• Blood glucose decreased to 250 mg/dL<br>• Alert and oriented x 3<br>• Preventing complications of treatment and immobility | **Expected Outcomes**<br>• Blood glucose <200 mg/dL<br>• Tolerating oral fluids and foods<br>• Tolerating activity<br>• Preventing complications of treatment and immobility | **Expected Outcomes**<br>• Demonstrates satisfactory knowledge of treatment, sick day management, DKA prevention, signs and symptoms of hypoglycemia and blood glucose monitoring<br>• Discharge without complications |

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## POST-TEST

This program can also be completed online at *www.MedEdToday.com*. Please provide 1 answer for each question (10 correct answers are necessary for successful completion). Place all answers on the exam answer key provided on the evaluation form following this post-test.

**1. Diabetic ketoacidosis is characterized by _____.**

a. Plasma glucose >250 mg/dL
b. Ketosis
c. Acidosis
d. Lipolysis
e. All of the above

**2. All of the following EXCEPT _____ are major precipitating causes of DKA.**

a. Infection
b. Hypoglycemia
c. Pancreatitis
d. Trauma
e. Myocardial infarction

**3. DKA occurs in _____ of patients with newly diagnosed diabetes.**

a. 5%–10%
b. 10%–20%
c. 20%–30%
d. 30%–40%
e. 40%–50%

**4. Which of the following generally differentiates HHS from DKA?**

a. Onset of DKA is more rapid than HHS
b. Minimal or no increase in lipolysis
c. Severity of ketosis
d. pH may be within normal limits
e. All of the above

**5. Vital signs in DKA typically reveal _____.**

a. Hypertension, bradycardia, and normal respiration
b. Orthostatic hypotension, bradycardia, and tachypnea
c. Orthostatic hypotension, tachycardia, and tachypnea
d. Hypertension, palpitation, and tachypnea

**6. Sodium bicarbonate therapy for DKA _____.**

a. Is not recommended when pH is ≥7.1
b. Can exacerbate intracellular acidosis
c. Can cause hypoglycemia
d. Both a & b

**7. Which of the following is a FALSE statement?**

a. DKA never occurs in type 2 diabetes.
b. Hyperventilation is a consequence of acidosis.
c. Stress associated with intercurrent illnesses may increase the body's requirement for insulin.
d. Diabetic neuropathy can result in the absence or delay of signs and symptoms of a myocardial infarction.

**8. Which action is not recommended for a patient with diabetes during sick days?**

a. Stop taking insulin when not eating.
b. Report infections to the physician.
c. Perform frequent tests of ketones.
d. Keep well hydrated.

**9. Rapid-acting insulin analogs may provide an alternative to intravenous regular human insulin for treating _____.**

a. Mild to moderate DKA
b. DKA of any severity
c. Hypoglycemic coma
d. Cerebral edema
e. None of the above

**10. How does the body compensate for acidosis?**

a. Renal elimination of ketones
b. Bicarbonate buffering
c. Retention of carbon dioxide
d. a and b
e. All of the above

**11. All of the following are objectives of the healthcare team for patients who have diabetes except _____.**

a. Teach patients to recognize the early signs of DKA
b. Teach patients to perform self-monitoring of ketones
c. Teach patients to omit their insulin dose during sick days
d. Identify risk factors that may require special intervention

**12. Potential complications during treatment for DKA include _____.**

a. Hypoglycemia
b. Hypochloremia
c. Schizophrenia
d. All of the above

**13. Which of the following statements is(are) true about DKA during pregnancy?**

a. It occurs more commonly during the first trimester.
b. An episode lasting 2 to 5 hours or longer can cause fetal death.
c. Fetal loss occurs in 20% of cases.
d. All of the above

**14. Special considerations are required for management of DKA in the elderly because _____.**

a. There is a high mortality rate in this population
b. Increased risk of fluid retention
c. Increased risk of thromboembolic events
d. All of the above

# EVALUATION FORM

## A Comprehensive Review of Diabetic Ketoacidosis and Hyperosmolar Hyperglycemic State in Adults
## Project ID: 3619 ES 13

Postgraduate Institute for Medicine (PIM) respects and appreciates your opinions. To assist us in evaluating the effectiveness of this activity and to make recommendations for future educational offerings, please take a few minutes to complete this evaluation form.

***You must complete this evaluation form to receive acknowledgment of participation for this activity.***

---

**Please answer the following questions by circling the appropriate rating:**

5 = Outstanding 4 = Good 3 = Satisfactory 2 = Fair 1 = Poor

**Extent to Which Program Activities Met the Identified Purpose**

| | | | | | |
|---|---|---|---|---|---|
| Provide clinicians treating diabetic patients with the latest information about the pathophysiology, diagnosis, and management of DKA and HHS to help reduce the incidence, morbidity, and mortality of the disorder. | 5 | 4 | 3 | 2 | 1 |

**Extent to Which Program Activities Met the Identified Objectives**

*Upon completion of this activity, participants should be better able to:*

| | | | | | |
|---|---|---|---|---|---|
| • Differentiate between DKA and HHS | 5 | 4 | 3 | 2 | 1 |
| • Describe the epidemiology and pathophysiology of DKA | 5 | 4 | 3 | 2 | 1 |
| • Identify the signs, symptoms, and complications of DKA | 5 | 4 | 3 | 2 | 1 |
| • Discuss methods of treatment and recovery care for DKA | 5 | 4 | 3 | 2 | 1 |
| • Describe potential complications during treatment of DKA and special considerations for pregnant and elderly individuals | 5 | 4 | 3 | 2 | 1 |
| • Provide strategies for the prevention of DKA | 5 | 4 | 3 | 2 | 1 |

**Overall Effectiveness of the Activity**

| | | | | | |
|---|---|---|---|---|---|
| • Was timely and will influence how I practice | 5 | 4 | 3 | 2 | 1 |
| • Will assist me in improving patient care | 5 | 4 | 3 | 2 | 1 |
| • Fulfilled my educational needs | 5 | 4 | 3 | 2 | 1 |
| • Avoided commercial bias or influence | 5 | 4 | 3 | 2 | 1 |

**Impact of the Activity**

*The information presented* (check all that apply)*:*

❏ Reinforced my current practice/treatment habits
❏ Provided new ideas or information I expect to use
❏ Will improve my practice/patient outcomes
❏ Enhanced my current knowledge base

Will the information presented cause you to make any changes in your practice? ❏ Yes ❏ No

If yes, please describe any change(s) you plan to make in your practice as a result of this activity:

How committed are you to making these changes? 5 (Very committed) 4 3 2 1 (Not at all committed)

**Future Activities**

Do you feel future activities on this subject matter are necessary and/or important to your practice?

❑ Yes ❑ No

Please list any other topics that would be of interest to you for future educational activities:

_____

_____

**Follow-up**

As part of our ongoing continuous quality-improvement effort, we conduct post-activity follow-up surveys to assess the impact of our educational interventions on professional practice. Please indicate your willingness to participate in such a survey:

❑ Yes, I would be interested in participating in a follow-up survey.

❑ No, I'm not interested in participating in a follow-up survey.

Additional comments about this activity:

_____

_____

_____

***If you wish to receive acknowledgment of participation for this activity, please complete the post-test by selecting the best answer to each question, complete this evaluation verification of participation and mail to: PIM, 367 Inverness Parkway, Suite 215, Englewood, CO 80112 or FAX to: 303-790-4876.***

**Post-test Answer Key**

| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|
| | | | | | | |
| 8 | 9 | 10 | 11 | 12 | 13 | 14 |
| | | | | | | |

**Request for Credit**

Name _____ Degree _____

Organization _____ Specialty _____

Address _____

City, State, Zip _____

Telephone _____ Fax _____ E-Mail _____

I certify my actual time to complete this educational activity to be:

❑ I participated in the entire activity and claim 1.5 credits.

❑ I participated in only part of the activity and claim _____ credits.

Signature _____ Date Completed _____