Diabetic Ketoacidosis

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A diagnosis of diabetic ketoacidosis requires the patient’s plasma glucose concentration to be above 250 mg per dL (although it usually is much higher), the pH level to be less than 7.30, and the bicarbonate level to be 18 mEq per L or less. Beta-hydroxybutyrate is a better measurement of the degree of ketosis than serum ketones. Intravenous insulin and fluid replacement are the mainstays of therapy, with careful monitoring of potassium levels. Phosphorous and magnesium also may need to be replaced. Bicarbonate therapy rarely is needed. Infection, insulin omission, and other problems that may have precipitated ketoacidosis should be treated. Myocardial infarction is a precipitating cause of diabetic ketoacidosis that is especially important to look for in older patients with diabetes. Cerebral edema is a major complication that occurs primarily in children. Education to prevent recurrence should be offered to all patients, including how to manage sick days and when to call a physician. (Am Fam Physician 2005;71:1705-14, 1721-2. Copyright© 2005 American Academy of Family Physicians.)

Many patients with diabetes die from diabetic ketoacidosis (DKA) every year. DKA is caused by reduced insulin levels, decreased glucose use, and increased gluconeogenesis from elevated counter regulatory hormones, including catecholamines, glucagon, and cortisol. DKA primarily affects patients with type 1 diabetes, but also may occur in patients with type 2 diabetes, and is most often caused by omission of treatment, infection, or alcohol abuse.1 Use of a standard protocol provides consistent results in treating DKA.2 An evidence-based guideline for the management of DKA from the American Diabetes Association (ADA) is the basis for much of this article.3

Initial Evaluation

Initial evaluation of patients with DKA includes diagnosis and treatment of precipitating factors (Table 14-18). The most common precipitating factor is infection, followed by noncompliance with insulin therapy.3 While insulin pump therapy has been implicated as a risk factor for DKA in the past, most recent studies show that with proper education and practice using the pump, the frequency of DKA is the same for patients on pump and injection therapy.19

DIFFERENTIAL DIAGNOSIS

Three key features of diabetic acidosis are hyperglycemia, ketosis, and acidosis. The conditions that cause these metabolic abnormalities overlap. The primary differential diagnosis for hyperglycemia is hyperosmolar hyperglycemic state (Table 23,20), which is discussed in the Stoner article21 on page 1723 of this issue. Common problems that produce ketosis include alcoholism and starvation. Metabolic states in which acidosis is predominant include lactic acidosis and ingestion of drugs such as salicylates and methanol.

Abdominal pain may be a symptom of ketoacidosis or part of the inciting cause of DKA, such as appendicitis or cholecystitis. If surgery is necessary, the timing needs to be individualized for each patient with input from a surgical consultant.

SIGNS AND SYMPTOMS

DKA can develop in less than 24 hours.3 Metabolic changes occur one and one half to two hours earlier in patients who are managed only with a short-acting insulin such as
Patients with DKA usually present with polyuria, polydipsia, polyphagia, weakness, and Kussmaul’s respirations. Nausea and vomiting are present in 50 to 80 percent of patients, and abdominal pain is present in about 30 percent. Coffee-ground emesis, usually from hemorrhagic gastritis, occurs in about 25 percent of vomiting patients. Often, the patient’s breath will have a fruity odor.

Body temperature usually is normal or low, even with an infection. If the patient’s temperature is elevated, infection invariably is present. Signs of dehydration, such as dry mucous membranes, tachycardia, and hypotension, often are found. Most patients are about 10 percent dehydrated. Consciousness ranges from alert to confused to a comatose state in less than 20 percent of patients.

**LABORATORY EVALUATION**

A standard laboratory work-up is listed in Table 3. The severity of DKA is determined primarily by the pH level, bicarbonate level, and mental status, and not by the blood glucose measurement. Although the bicarbonate level typically is low, it may be normal or high in patients with vomiting, diuretic use, or alkali ingestion. If the serum osmolality is less than 320 mOsm per kg, etiologies other than DKA should be considered. Osmolality can be calculated using the formula for effective osmolality (mOsm per kg):

\[
2 \times \text{Na}^+ \text{ (mEq per L)} + \text{plasma glucose (mg per dL)}
\]

In this equation, Na\(^+\) is the serum sodium level. Although potassium is included in some formulas, it is not included in the formula recommended by the ADA. Blood urea nitrogen is not included in this measurement because urea has less osmotic activity.

Beta-hydroxybutyrate accounts for about 75 percent of ketones in ketoacidosis, and when available it is preferred for monitoring DKA over the nitroprusside method, which only measures acetoacetate. A value greater than 3 mg per dL is considered abnormal. The beta-hydroxybutyrate level may not normalize during the first one to two days of treatment. Although it is not monitored routinely during treatment, the beta-hydroxybutyrate level usually is less than 1.5 mg per dL after the first 12 to 24 hours of treatment.

Liver enzymes also are elevated frequently in patients...
Diabetic Ketoacidosis

with DKA because of unknown causes.26 If pancreatitis is suspected, it must be diagnosed clinically. In one study10 of ketoacidosis, amylase was elevated in 21 percent and lipase in 29 percent of patients. If pancreatitis is suspected, contrast-enhanced computed tomography (CT) may be useful for diagnosis in selected patients. If the patient has significant hypertriglyceridemia, it can falsely lower glucose and sodium measurements by dilution. Leukocytosis may be present in DKA without infection.

Treatment

A priority of treatment should be to protect and maintain the airway, particularly in the obtunded patient, and to treat shock if present. Patients should be monitored closely and frequently. Blood glucose should be evaluated every one to two hours until the patient is stable, and the blood urea nitrogen, serum creatinine, sodium, potassium, and bicarbonate levels should be monitored every two to six hours depending on the severity of DKA.3 Cardiac monitoring may be warranted for patients with significant electrolyte disturbances. Treatment also should be directed at the underlying cause of the DKA, including antibiotics for suspected or identified infection. Although it is important to monitor urinary output, urinary catheterization is not advised routinely.

INPATIENT VS. OUTPATIENT TREATMENT

Selected patients with mild DKA who are alert and taking fluids orally may be treated under observation and sent home without admission.3 The ADA admission guidelines are a plasma glucose concentration greater than 250 mg per dL (13.9 mmol per L) with an arterial pH level

TABLE 1
Causes of Diabetic Ketoacidosis

<table>
<thead>
<tr>
<th>Common causes by frequency</th>
<th>Other causes</th>
<th>Selected drugs that may contribute to diabetic ketoacidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infection, particularly pneumonia, urinary tract infection, and sepsis4</td>
<td>Acanthosis nigricans6</td>
<td>Atypical antipsychotic agents12</td>
</tr>
<tr>
<td>Inadequate insulin treatment or noncompliance4</td>
<td>Acromegaly7</td>
<td>Corticosteroids13</td>
</tr>
<tr>
<td>New-onset diabetes4</td>
<td>Arterial thrombosis, including mesenteric and iliac5</td>
<td>FK50614</td>
</tr>
<tr>
<td>Cardiovascular disease, particularly myocardial infarction5</td>
<td>Cerebrovascular accident5</td>
<td>Glucagon15</td>
</tr>
<tr>
<td></td>
<td>Hemochromatosis6</td>
<td>Interferon16</td>
</tr>
<tr>
<td></td>
<td>Hyperthyroidism9</td>
<td>Sympathomimetic agents including albuterol (Ventolin), dopamine (Intropin), dobutamine (Dobutrex), terbutaline (Bricanyl),17 and ritodrine (Yutopar)18</td>
</tr>
</tbody>
</table>

Information from references 4 through 18.

TABLE 2
Diagnostic Criteria for Diabetic Ketoacidosis and Hyperosmolar Hyperglycemic State

<table>
<thead>
<tr>
<th>Mild DKA</th>
<th>Moderate DKA</th>
<th>Severe DKA</th>
<th>HHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma glucose (mg per dL [mmol per L])</td>
<td>&gt; 250 (13.9)</td>
<td>&gt; 250</td>
<td>&gt; 250</td>
</tr>
<tr>
<td>Arterial pH</td>
<td>7.25 to 7.30</td>
<td>7.00 to 7.24</td>
<td>&lt; 7.00</td>
</tr>
<tr>
<td>Serum bicarbonate (mEq per L)</td>
<td>15 to 18</td>
<td>10 to &lt; 15</td>
<td>&lt; 10</td>
</tr>
<tr>
<td>Urine ketones</td>
<td>Positive</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>Serum ketones</td>
<td>Positive</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>Beta-hydroxybutyrate</td>
<td>High</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Effective serum osmolality (mOsm per kg)†</td>
<td>Variable</td>
<td>Variable</td>
<td>Variable</td>
</tr>
<tr>
<td>Anion gap†</td>
<td>&gt; 10</td>
<td>&gt; 12</td>
<td>&gt; 12</td>
</tr>
<tr>
<td>Alteration in sensoria or mental obtundation</td>
<td>Alert</td>
<td>Alert/drowsy</td>
<td>Stupor/coma</td>
</tr>
</tbody>
</table>

DKA = diabetic ketoacidosis; HHS = hyperosmolar hyperglycemic state.

*—Effective serum osmolality = 2 × measured Na (mEq per L) + (glucose [mg per dL] + 18).
†—Anion gap = Na+ – (Cl− + HCO3−) (mEq per L).

below 7.30, a serum bicarbonate level of less than 15 mEq per L, and a moderate or greater level of ketones in the serum or urine.\textsuperscript{27} Patients with severe DKA should be admitted to the intensive care unit.

**FLUIDS**

Fluid deficits are typically 100 mL per kg of body weight.\textsuperscript{3} Fluid replacement alone will lower blood glucose. Tracer studies have found that during the first four hours of therapy for DKA, up to 80 percent of the decline in glucose concentration may be caused by rehydration.\textsuperscript{28}

Fluid guidelines are summarized on the flowchart in Figure 1.\textsuperscript{3} When giving fluids, the average rate of change in effective serum osmolality ideally should not be more than 3 mOsm per hour. Patients who are able to drink can take some or all of their fluid replacement orally. Fluid intake should be modified based on urinary output. Urinary output will decrease as the osmotic diuretic effect of hyperglycemia is reduced.

When the blood glucose level has dropped below 250 mg per dL, the patient may be given fluid with 5 percent dextrose, such as 0.45 normal saline. If dextrose is not given, further ketosis may occur.

**INSULIN**

An intravenous insulin drip is the current standard of care for diabetic ketoacidosis, primarily because of the more rapid onset of action. Studies\textsuperscript{29} comparing intravenous insulin with subcutaneous or intramuscular insulin have found a quicker decrease in glucose and ketone levels, but no improvement in morbidity and mortality. Insulin may be mixed in a standard concentration of 1 U per 10 mL of normal saline. Common adult rates are 5 to 7 U per hour. A standard regimen is given in Figure 1.\textsuperscript{3} When the blood glucose level is less than 250 mg per dL, the intravenous insulin rate usually is decreased, or the patient is switched to subcutaneous insulin to maintain plasma glucose in the range of 150 to 200 mg per dL (8.3 to 11.1 mmol per L) until metabolic control is achieved.

Regular insulin should be used intravenously. Lispro and aspart (NovoLog) insulin are more expensive and do not work faster than regular insulin when given intravenously. A newly published regimen is treatment of DKA with subcutaneous aspart or lispro insulin.\textsuperscript{29,30} In one study,\textsuperscript{30} patients who were medically stable after initial fluid resuscitation were treated with a loading dose of 0.3 U per kg of aspart insulin, followed by 0.1 U per kg every hour. There were no significant differences in outcomes between the aspart and intravenous insulin regimens. A similar study\textsuperscript{29} comparing subcutaneous lispro insulin in a medical ward with an intravenous insulin drip in the intensive care unit showed similar outcomes, except for a 40 percent reduction in cost for patients treated in the medical ward. Long-acting insulin normally is stopped during treatment of DKA. If the patient is on an insulin pump, it should be stopped, and the patient should be switched to an intravenous infusion.\textsuperscript{31}

If an intravenous infusion pump is not available, insulin can be given intramuscularly. Insulin is absorbed more rapidly intramuscularly than if given subcutaneously.\textsuperscript{32} A regimen for intramuscular insulin is given in Figure 1.\textsuperscript{3} This regimen advises that an initial dose of insulin be given intravenously and intramuscularly. When intravenous access is unavailable, studies have found that giving the entire initial dose intramuscularly also is effective.\textsuperscript{33} If intramuscular insulin is used, it is important to use a needle that is long enough to ensure that the insulin is not given subcutaneously.

**POTASSIUM**

Whole body potassium deficits typically are 3 to 5 mEq per L (3 to 5 mmol per L). Acidosis increases potassium levels and glucose administered with insulin lowers them. Before treatment of DKA, the level of potassium usually is normal or elevated. Potassium should be started as soon as adequate urine output is confirmed and the potassium level is less than 5 mEq per L.\textsuperscript{3} Usually 20 to 30 mEq (20 to 30 mmol) of potassium is given for each liter of fluid replacement. If the potassium level is less than 3.3 mEq per L (3.3 mmol per L), potassium replacement should be given immediately and insulin should be started only after the potassium level is above 3.3 mEq per L.\textsuperscript{3}

**BICARBONATE**

Studies of patients with a pH level of 6.9 or higher have found no evidence that bicarbonate is beneficial,\textsuperscript{34} and some studies have suggested bicarbonate therapy may be harmful for these patients.\textsuperscript{35-37} The flowchart in Figure 1 advises giving no bicarbonate if the pH level is greater than 6.9. Because there are no studies on patients with a pH level below 6.9, giving bicarbonate as an isotonic solution still is recommended. Bicarbonate therapy lowers potassium levels; therefore, potassium needs to be monitored carefully.

**PHOSPHATE**

Although the phosphate level frequently is low in patients with DKA, good-quality studies have shown that routine phosphate replacement does not improve outcomes in DKA, and excessive replacement can lead to hypocalcemia.\textsuperscript{3,36-40} If the patient’s serum phosphate level is below normal,
consider giving one third to one half of the potassium may be given in the form of potassium phosphate, provided the level of serum calcium is monitored closely.3,41

MAGNESIUM
A serum deficit of 1 to 2 mEq per L (0.50 to 1 mmol per L) of magnesium usually exists. In addition to alterations in magnesium metabolism from DKA, many patients with diabetes have taken medications such as diuretics that also may lower magnesium levels. Symptoms of magnesium deficiency are difficult to recognize and overlap with symptoms caused by deficiencies of calcium, potassium, and sodium. Paresthesias, tremor, carpopedal spasm, agitation, seizures, and cardiac dysrhythmias all are reported symptoms. Checking magnesium levels and correcting low levels should be considered in patients with DKA. Patients usually are symptomatic at serum levels of 1.2 mg per dL (0.50 mmol per L) or lower.42 If the level is below normal (i.e., less than 1.8 mg per dL [0.74 mmol per L]) and symptoms are present, administration of magnesium should be considered.42

SODIUM
Whole body sodium deficits typically are 7 to 10 mEq per L (7 to 10 mmol per L). Serum sodium is falsely lowered by 1.6 mEq for every 100 mg per dL increase in blood glucose. Hyponatremia needs to be corrected only when the sodium level is still low after adjusting for this effect. For example, in a patient with a serum glucose concentration of 600 mg per dL (33.3 mmol per L) and a measured serum sodium level of 130, the true serum sodium level is 130 + (1.6 × 5) = 138. A high serum sodium level almost always indicates hypernatremic dehydration.

Complications
Common complications of DKA include hypoglycemia, hypokalemia, and recurrent hyperglycemia. These may be minimized by careful monitoring. Hyperchloremia is a common but transient finding that usually requires no special treatment.

Cerebral edema is a rare but important complication of DKA. Although it can affect adults, it is more common in young patients, occurring in 0.7 to 1.0 percent of children with DKA.3 Early signs of cerebral edema include headache, confusion, and lethargy. Papilledema, hypertension, hyperpyrexia, and diabetes insipidus also may occur. Patients typically improve mentally with initial treatment of DKA, but then suddenly worsen. Dilated ventricles may be found on CT or magnetic resonance imaging. Treatment of suspected cerebral edema should not be delayed for these tests to be completed. In more severe cases, seizures, pupillary changes, and respiratory arrest with brain-stem herniation may occur. Once severe symptoms occur, the mortality rate is greater than 70 percent, and only about 10 percent of patients recover without sequelae.3

Avoiding overhydration and limiting the rate at which the blood glucose level drops may reduce the chance of cerebral edema.3 However, some patients may present with cerebral edema before treatment is started. About 10 percent of the patients initially diagnosed with cerebral edema have other intracranial pathology such as subarachnoid hemorrhage.43 Mannitol (Osmitrol) therapy and hyperventilation have been recommended based on limited evidence.44,45

Special Situations—Young and Old Patients
The main differences in the management of children and adolescents compared with adults are the greater care in administering electrolytes, fluids, and insulin based on

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**TABLE 3**

<table>
<thead>
<tr>
<th>Standard Laboratory Assessment for Patients with Diabetic Ketoacidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma glucose</td>
</tr>
<tr>
<td>Electrolytes with calculated anion gap and effective osmolality</td>
</tr>
<tr>
<td>Phosphorous</td>
</tr>
<tr>
<td>Blood urea nitrogen and creatinine</td>
</tr>
<tr>
<td>Beta-hydroxybutyrate or serum ketones if not available</td>
</tr>
<tr>
<td>Complete urinalysis with urine ketones by dipstick</td>
</tr>
<tr>
<td>Arterial blood gas or venous pH level if not available</td>
</tr>
<tr>
<td>Complete blood count with differential</td>
</tr>
<tr>
<td>Electrocardiography</td>
</tr>
<tr>
<td><strong>As indicated</strong></td>
</tr>
<tr>
<td>Bacterial cultures of urine, blood, throat, or other sites of suspected infection</td>
</tr>
<tr>
<td>Chest radiography if pneumonia or cardiopulmonary disease is suspected</td>
</tr>
<tr>
<td>Magnesium if patient has signs of hypomagnesemia such as cardiac arrhythmias, is alcoholic, or is taking diuretics</td>
</tr>
<tr>
<td>A1C level may help determine whether this is an acute episode in a patient with well-controlled, undiagnosed, or poorly controlled diabetes.</td>
</tr>
</tbody>
</table>

the weight of the patient and increased concern about high fluid rates inducing cerebral edema. A flowchart for the management of DKA in children and adolescents from the ADA guideline is shown in Figure 2. A growing problem is the development of type 2 diabetes in obese children. Although DKA is less common in these patients than among those with type 1 diabetes, it does occur. C-peptide levels may be helpful for determining the type of diabetes and guiding subsequent treatment. Risk factors for adolescent type 2 diabetes are hypertension and acanthosis nigricans.

Older patients are less likely to be on insulin before developing DKA, less likely to have had a previous episode of DKA, typically require more insulin to treat the DKA, have

**Management of Adults with Diabetic Ketoacidosis**

Perform history and physical examination, order laboratory tests, and evaluate severity of diabetic ketoacidosis. Quickly start 0.9 percent NaCl at 1.0 L per hour (15 to 20 mL per kg) for first hour.

- **IV fluids after first hour**
  - **Determine hydration status.**
    - **Hypovolemic shock**
      - Administer 0.9 percent NaCl 1.0 L per hour until shock corrected.
    - **Mild hypotension or normal**
      - Determine corrected serum sodium level.
    - **Cardiogenic shock**
      - Consider administering fluids based on hemodynamic monitoring.
  - **Determine corrected serum sodium level.**
    - **High or normal**
      - Give 0.45 percent NaCl at 4 to 14 mL per kg per hour depending on hydration.
    - **Low**
      - Give 0.9 percent NaCl at 4 to 14 mL per kg per hour depending on hydration.
  - **When serum glucose is 250 mg per dL or less, change to 5 percent dextrose with 0.45 percent NaCl at 150 to 250 mL per hour until metabolic control is achieved.**

- **Insulin (hold until potassium is 3.3 mEq per L [3.3 mmol per L] or greater)**
  - **IV**
    - **IV bolus of regular insulin 0.15 units per kg**
      - 0.1 units per kg per hour IV insulin infusion
    - **IV bolus of regular insulin 0.2 units per kg plus 0.2 units per kg IM or SC**
      - If serum glucose does not fall by 50 to 70 mg per dL per hour
        - Double insulin infusion hourly until glucose falls by 50 to 70 mg per dL.
        - Give hourly IV insulin bolus of 10 units until glucose falls by 50 to 70 mg per dL.
    - **Give hourly IV insulin bolus of 10 units until glucose falls by 50 to 70 mg per dL.**
      - When serum glucose is 250 mg per dL or less, continue IV infusion of 0.05 to 0.10 per kg per hour or give 5 to 10 units every two hours to keep serum glucose between 150 and 200 mg per dL until metabolic control is achieved.

**Figure 1.** Algorithm for the management of adults with diabetic ketoacidosis. (NaCl = sodium chloride; IM = intramuscular; IV = intravenous; SC = subcutaneous.)
Diabetic Ketoacidosis

Management of Patients Younger than 20 Years with Diabetic Ketoacidosis* or Hyperosmolar Hyperglycemic State†

Complete initial evaluation.‡ Start IV fluids: 10 to 20 mL per kg, 0.9 percent NaCl in the initial hour.

IV fluids

Determine hydration status.

Hypovolemic shock

Administer 0.9 percent NaCl (20 mL per kg per hour) and/or plasma expander until shock resolved.

Mild hypotension

Administer 0.9 percent NaCl (10 mL per kg per hour) for initial hour.

Serum glucose reaches 250 mg per dL

Change to 5 percent dextrose with 0.45 to 0.75 percent NaCl, at a rate to complete rehydration in 48 hours and to maintain glucose between 150 and 250 mg per dL (10 percent dextrose with electrolytes may be required).

Check glucose and electrolyte levels every two to four hours until stable. Look for precipitating causes. After resolution of diabetic ketoacidosis, initiate SC insulin (0.5 to 1.0 U per kg per day given as 2/3 in the morning [1/3 short-acting, 2/3 intermediate-acting], 1/3 in the evening [1/2 short-acting, 1/2 intermediate-acting]), or as 0.1 to 0.25 U per kg regular insulin every six to eight hours during the first 24 hours for new patients to determine insulin requirements.

< 2.5 mEq per L (2.5 mmol per L)

2.5 to 3.5 mEq per L (3.5 mmol per L)

3.5 to 5.0 mEq per L (5.0 mmol per L)

> 5.0 mEq per L (5.0 mmol per L)

Administer potassium 40 to 60 mEq per L (40 to 60 mmol per L) in IV solution|| until > 3.5 mEq per L. Monitor potassium hourly.

Continue as above.

Administer potassium 30 to 40 mEq per L in IV solution|| to maintain serum potassium at 3.5 to 5.0 mEq per L.

Administer 1 mEq per kg of potassium chloride in IV over one hour. Withhold insulin until potassium > 2.5 mEq per L.

< 2.5 mEq per L

2.5 to 3.5 mEq per L

> 3.5 mEq per L

Administer potassium 0.1 U per kg IV bolus followed by 0.1 U per kg per hour SC or IM.

Continue until acidosis clears (pH > 7.3, bicarbonate > 15).

Decrease to 0.05 U per kg per hour until SC insulin replacement initiated.

Check results of hourly potassium monitoring.

pH < 7.0 after initial hour of hydration?

Continue as above.

Administer potassium 30 to 40 mEq per L in IV solution|| to maintain serum potassium at 3.5 to 5.0 mEq per L.

No bicarbonate indicated.

Over one hour, administer sodium bicarbonate (2 mEq per kg) added to NaCl to produce a solution that does not exceed 155 mEq per L (155 mmol per L) of sodium over one hour.

< 2.5 mEq per L

2.5 to 3.5 mEq per L

> 3.5 mEq per L

No

Yes

< 2.5 mEq per L

2.5 to 3.5 mEq per L

> 3.5 mEq per L

Administer potassium 40 to 60 mEq per L (40 to 60 mmol per L) in IV solution|| until > 3.5 mEq per L. Monitor potassium hourly.

Do not give IV potassium. Monitor potassium hourly until < 5.0 mEq per L.

Potassium

Assess need for bicarbonate.

Do not give IV potassium.

Monitor potassium hourly until < 5.0 mEq per L.

Repeat pH after initial hydration bolus.

Continue until acidosis clears (pH > 7.3, bicarbonate > 15).


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*—Diagnostic criteria: blood glucose > 250 mg per dL, venous pH < 7.3, bicarbonate < 15 mEq per L, moderate ketonuria or ketonemia.
†—Diagnostic criteria: blood glucose > 600 mg per dL, venous pH > 7.3, bicarbonate > 15 mEq per L, and altered mental status or severe dehydration.
‡—After the initial history and physical examination, immediately obtain blood glucose, venous blood gases, electrolytes, blood urea nitrogen, creatinine, calcium, phosphorus, and urine analysis.
§—Usually 1.5 times the 24-hour maintenance requirements (about mL per kg per 1 per hour–1) will accomplish a smooth rehydration; do not exceed two times the maintenance requirement.
||—The potassium in solution should be 1/3 potassium phosphate and 2/3 potassium chloride or Kacetate.
Diabetic Ketoacidosis

### TABLE 4

**Strategies to Prevent Diabetic Ketoacidosis**

<table>
<thead>
<tr>
<th>Strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetic education</td>
</tr>
<tr>
<td>Blood glucose monitoring</td>
</tr>
<tr>
<td>Sick-day management</td>
</tr>
<tr>
<td>Home monitoring of ketones or beta-hydroxybutyrate</td>
</tr>
<tr>
<td>Supplemental short-acting insulin regimens</td>
</tr>
<tr>
<td>Easily digestible liquid diets when sick</td>
</tr>
<tr>
<td>Reducing, rather than eliminating, insulin when patients are not eating</td>
</tr>
<tr>
<td>Guidelines for when patients should seek medical attention</td>
</tr>
<tr>
<td>Case monitoring of high-risk patients</td>
</tr>
<tr>
<td>Special education for patients on pump management</td>
</tr>
</tbody>
</table>

Information from references 49 through 51.

a standard subcutaneous insulin regimen by injection or insulin pump should be started.

Intravenous insulin should continue for one to two hours after initiation of subcutaneous insulin. For patients who are unable to eat, intravenous insulin may be continued to maintain the blood glucose in a target range (i.e., 80 to 140 mg per dL [4.4 to 7.8 mmol per L]).

Prevention of another episode should be part of the treatment of DKA. Most patients with DKA will need lifetime insulin therapy after discharge from the hospital. Education about diabetes is a cornerstone of prevention that also has been found to reduce length of stay.48 Strategies for prevention are listed in Table 4.49-51

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### REFERENCES

Diabetic Ketoacidosis


