Diabetic ketoacidosis (DKA) is a common endocrine emergency in veterinary medicine. DKA results in acute metabolic derangement in dogs and cats, requiring prompt medical treatment.

The nursing management approach to patients with DKA is similar to that of patients with other serious illnesses. Veterinary technicians must have a basic understanding of the disease as well as the ability to perform various diagnostic and therapeutic procedures. Knowledge of the disease process and possible outcomes will enable technicians to anticipate and identify potential complications.

Pathophysiology

Diabetic ketoacidosis, which is a life-threatening complication of diabetes mellitus (DM), is characterized by hyperglycemia, ketosis, acidosis, dehydration, and electrolyte imbalances. Hyperglycemia and ketoacidosis occur during insulin deficiency or resistance. Consequently, glucose and ketoacids are overproduced and underused. Despite the onset of hyperglycemia, cellular starvation occurs. Cell energy requirements that are not met stimulate gluconeogenesis (see the Glossary) and increased hepatic glycogenolysis. As a result, the animal’s body breaks down fat and protein stores to meet the energy requirements of cellular metabolism. Because the body is unable to keep up with the rate of production of this alternative energy source, ketone bodies accumulate in the blood.

Eventually, hyperglycemia exceeds the renal threshold for glucose resorption and glucosuria, causing osmotic diuresis. Osmotic diuresis results in losses of sodium, potassium, phosphorus, magnesium, and body water. Increased water loss leads to severe dehydration and hypovolemia. Nausea, anorexia, and vomiting occur when ketonemia and hyperglycemia stimulate the chemoreceptor trigger zone, further contributing to dehydration. Dehydration decreases tissue perfusion, causing lactic acid production and exacerbating the existing acidosis (Figure 1).

Assessment

History and Risk Factors

Although DKA occurs mostly in dogs or cats with previously undiagnosed DM, the condition may also develop in previously diagnosed diabetics with the same predisposing factors or in animals in which insulin has been administered improperly. Veterinary patients may have a history of polydipsia, polyuria, and weight loss. The acute presentation may include the sudden onset of anorexia, depression, abdominal pain, weakness, and vomiting. Many of the precipitating factors seen in humans may also contribute to DKA in animals. These risk fac-
tors may include too little insulin, infection, severe stress, hypokalemia, renal failure, inadequate fluid intake, and ingestion of drugs that decrease insulin secretion (e.g., β-blockers, thiazides) or cause insulin resistance (e.g., glucocorticoids, progestational agents). Many disorders have been reported to coexist with DKA, including Cushing’s syndrome, inflammatory bowel disease, pancreatitis, pneumonia, pyoderma, pyometra, renal insufficiency, and urinary tract infection.

**Physical Examination**

The initial physical examination should focus on hydration status, central nervous system involvement and/or depression, and any coexistent conditions. Physical findings may include dehydration, depression, weakness, tachycardia, and hypotension. Affected animals may be tachypneic or may experience slow, deep breathing patterns (i.e., Kussmaul respiration). Patients may also have an acetone odor on the breath.

**Laboratory Diagnostics**

Laboratory diagnostics (e.g., blood and urine glucose levels) will be needed to confirm or support a tentative diagnosis of DKA as well as to monitor therapy. Blood glucose (BG) values in dogs and cats have ranged from 200 to over 1000 mg/dl (mean, 500 mg/dl). Ketonuria or ketonemia should be documented to differentiate DKA from uncomplicated DM. Electrolyte (e.g., sodium, potassium, chloride, magnesium) and acid–base (e.g., blood gases, total CO$_2$) disorders, which are major components of the disease process, should also be monitored. A complete blood count, serum chemistry profile, urinalysis, and urine culture should be conducted to determine any coexistent problems.

**Nursing Management**

The goals of therapy in DKA are to correct dehydration, restore intravascular volume, normalize BG levels, and correct electrolyte and acid–base abnormalities.

**Restoring Fluid Volume Deficit**

The fluid volume deficit results from decreased circulating volume secondary to hyperglycemia and its induced...
osmotic diuresis. Fluid loss may also be caused by vomiting and lack of fluid intake. Within 20 to 24 hours after initiating fluid therapy, the goal is to return the patient to a normovolemic state evidenced by normal blood pressure; normal heart rate; normal central venous pressure; balanced ins and outs; sufficient urine production; normal skin turgor; and pink, moist mucous membranes.

A central venous catheter should be placed so that periodic blood samples can be easily obtained and central venous pressure measurements can be taken to help guide fluid therapy. Initially, the fluid type to be administered will be dictated by electrolyte status. Because many DKA patients also have hyponatremia, 0.9% saline is often given. DKA patients typically experience potassium depletion; therefore, fluids that are administered should be potassium enriched. Fluid rates and volumes will depend on the severity of dehydration, the animal’s maintenance needs, and abnormal ongoing losses (i.e., vomiting, diarrhea, diuresis). Caution should be exercised when giving hypotonic sodium solutions because these fluids have an increased amount of free water relative to isotonic solutions. Too much free water will put the patient at risk for developing cerebral edema. When the patient’s glucose declines to 250 mg/dl or less, 50% dextrose should be added to the fluid therapy to make a final dextrose concentration of 2.5% to 5%.

**Normalizing Blood Glucose Levels**

Regular crystalline insulin is recommended for treating DKA. Insulin therapy will decrease BG levels by driving glucose into the cells, thereby providing them with an alternative energy source other than ketone-producing fatty acids (see Effects of Insulin). Insulin therapy also drives potassium into the cells and will result in decreased serum potassium levels, thus unmasking a total body potassium deficit. Insulin protocols include intermittent intramuscular (IM) and continuous low-dose intravenous (IV) infusion techniques.

When using the IM technique, insulin should be administered hourly; the dose should be adjusted based on the rate of declining glucose levels. Once the animal’s BG level approaches 250 mg/dl, the hourly IM insulin dose should be administered subcutaneously every 4 to 6 hours. When the patient begins eating and drinking and is no longer ketogenic or vomiting, long-acting insulin may be used.

In the continuous low-dose IV infusion technique, the regular insulin dose should be diluted in 250 ml of 0.9% saline solution. The insulin infusion should be piggybacked (Figure 2) onto the primary fluid line and administered with a fluid infusion pump. Because insulin binds to glass and plastic, the first 50 ml of the infusion should be discarded.

Regardless of the insulin protocol used, serum glucose levels should not be allowed to drop too fast; otherwise, the patient is at risk for developing cerebral edema. Cerebral edema occurs when an osmotic gradient develops between the brain and the extravascular fluid space.

In addition to insulin, fluid therapy can be used to reduce serum glucose concentration. Fluids enhance glucose excretion by increasing glomerular filtration and urine flow. Fluids also decrease the secretion of diabetogenic hormones (i.e., epinephrine, norepinephrine, cortisol, glucagon). Diabetogenic hormones stimulate hyperglycemia.3

Initially, BG concentration should be monitored every 1 to 2 hours. BG concentration should decline by 50 to 100 mg/dl/hour.3 Patients should be monitored for hypoglycemia, which can be characterized by lethargy, depression, ataxia, weakness, seizures, or coma (see Insulin Overdose and Clinical Signs of Hypoglycemia). Therapy should include IV bolus administration of 0.25 g/kg (0.5 ml/kg) of 50% dextrose followed by a continuous infusion of 2.5% to 5% dextrose.

**Correcting Electrolyte and Acid–Base Abnormalities**

As mentioned, sodium loss through diuresis can be correct-
ed with fluid therapy. Although the animal’s serum potassium concentration may initially be decreased, normal, or elevated, the patient’s total body stores of potassium may actually be depleted. Insulin therapy will drive serum potassium into the cells, causing a lowering of serum potassium concentration and possibly the development of hypokalemia. Correction of dehydration and metabolic acidosis can also cause hypokalemia by dilutional effects and redistribution, respectively. Potassium supplementation will be needed; therefore, potassium chloride should be added to the fluids when serum potassium levels are known to be low (Table I).

If potassium levels are unknown, hypokalemia may be expected when the fluid deficit is caused by gastrointestinal loss, diuresis, and anorexia. Based on the magnitude of these losses, potassium depletion can be classified as mild, moderate, or severe; fluids should, therefore, be supplemented with 20, 30, or 40 mEq/L of potassium, respectively. If the patient is hypophosphatemic, deficit requirements of potassium and phosphorus may be replaced by fluid supplements consisting of divided doses of potassium phosphate and potassium chloride. Care should be exercised when administering potassium supplements. The maximum potassium administration rate is 0.5 mEq/kg/hour. Initially, electrolytes should be monitored every 2 to 4 hours. Ultimately, the patient’s condition will dictate the frequency of monitoring.

In the absence of electrolyte measurements, clinical signs and electrocardiographic monitoring may be used. Clinical signs of hypokalemia include severe muscle weakness, cervical ventroflexion, and arrhythmias. Clinical signs of hyperkalemia include bradycardia, weakness, and neuromuscular paralysis. Subtle electrocardiographic changes that are associated with these conditions may appear (see Electrocardiographic Changes That Occur with Hypokalemia and Hyperkalemia; Figure 3).

Ketones are acids that are buffered by bicarbonate in extracellular fluid. When patients experience excessive ketone production and bicarbonate deficits, metabolic acidosis develops. Decreased tissue perfusion may also contribute to this condition. Insulin and fluid therapy will help correct acid–base abnormalities. The use of sodium bicarbonate (NaHCO₃) to correct metabolic acidosis is considered controversial. Clear benefits of its use have not been demonstrated in humans with DKA. One study showed that NaHCO₃ may actually delay the resolution of ketosis with DKA. Theoretic arguments against the use of NaHCO₃ include the development of cerebral acidosis following NaHCO₃ administration. NaHCO₃ should be considered if the pH is less than 7.2, the base deficit is −10 mEq/L or larger, or the bicarbonate or total CO₂ is less than 12 mEq/L. When NaHCO₃ is administered, the dosing should be conservative. One option is to give enough NaHCO₃ over 30 minutes to correct the pH back to 7.2, base deficit to −10 mEq/L, or the bicarbonate/total CO₂ back to 12 mEq/L. Another option is to give half of the total base or serum bicarbonate deficit over 6 hours.

Resolution of the Diabetic Ketoacidosis

After DKA has been resolved and the patient’s condition is stable (eating and drinking, no vomiting), a longer-acting insulin preparation may be used. When the patient is discharged, the owner must notify veterinary personnel if the clinical signs of DM (i.e., polyuria, polydipsia, polyphagia, weight loss) or

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**TABLE I**

<table>
<thead>
<tr>
<th>Measured Concentration</th>
<th>Suggested Supplementation</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;5.5</td>
<td>0</td>
</tr>
<tr>
<td>3.5–5.5</td>
<td>5</td>
</tr>
<tr>
<td>3.0–3.4</td>
<td>20</td>
</tr>
<tr>
<td>2.5–2.9</td>
<td>30</td>
</tr>
<tr>
<td>2.0–2.4</td>
<td>40</td>
</tr>
<tr>
<td>&lt;2.0</td>
<td>50</td>
</tr>
</tbody>
</table>

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**Electrocardiographic Changes That Occur with Hypokalemia and Hyperkalemia**

- **Hypokalemia**
  - Prolonged Q-T interval
  - Depressed ST segment
  - Depressed T-wave amplitude
  - Appearance of U wave

- **Hyperkalemia**
  - Bradycardia
  - Decreased P-wave amplitude
  - Prolonged P-R interval
  - Prolonged QRS complex
  - Spiked T wave

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**Figure 3**—Electrocardiogram taken from a hyperkalemic (7.6 mmol/L) patient. Note the absence of P waves, a prolonged QRS interval, and a spiked T wave. (Copyright © Craig Cornell, RVT, VTS [ECC], Davis, CA)
hypoglycemia (i.e., lethargy, depression, ataxia, weakness, seizures, coma) recur. Owners should be instructed to monitor water intake, urine output, body weight, and appetite. When these factors are normal, diabetic patients are usually adequately controlled.

**Case Report**

Lulu, a 6-kg, 8-year-old spayed miniature poodle, presented with a 2-week history of vomiting that occurred two to three times per day for the first week. Although the poodle did not appear to be lethargic, it seemed to be getting progressively worse. The dog began losing its appetite 1 week earlier and became anorectic 4 days before admission. There was a history of polyuria and polydipsia before the onset of vomiting. Lulu was not on any medications, and the animal’s vaccinations were current.

On physical examination, the poodle was depressed but responsive. Temperature was 103.5°F, with a respiratory rate of 60 breaths/minute, a body condition score of 6 out of 9, decreased skin elasticity (about 8% dehydrated), and increased breath sounds in all lung fields. The midabdomen was painful; large, firm intestines were palpated with hepatomegaly present. The right prescapular lymph nodes were enlarged at 1 to 1.5 cm. The dog’s breath had an acetone smell.

**Initial Plan**

Based on history and physical examination findings, the veterinarian ordered a complete blood count to rule out inflammation and dehydration. A serum chemistry panel (including electrolytes) was conducted to assess metabolic and electrolyte de-

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**TABLE II**

**Lulu’s Abnormal Laboratory Values and Reference Ranges**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Lulu’s Values</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metamyelocytes</td>
<td>750/µl</td>
<td>0</td>
</tr>
<tr>
<td>Bands</td>
<td>9750/µl</td>
<td>0–300/µl</td>
</tr>
<tr>
<td>Leukocytes</td>
<td>37,500/µl</td>
<td>6000–17,000/µl</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>57%</td>
<td>37%–55%</td>
</tr>
<tr>
<td>Total protein</td>
<td>9.6 g/dl</td>
<td>6.0–8.0 g/dl</td>
</tr>
<tr>
<td>Serum glucose</td>
<td>741 mg/dl</td>
<td>70–120 mg/dl</td>
</tr>
<tr>
<td>Potassium</td>
<td>2.1 mmol/L</td>
<td>4.1–5.3 mmol/L</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>2.9 mg/dl</td>
<td>3.0–6.2 mg/dl</td>
</tr>
<tr>
<td>Total carbon dioxide</td>
<td>11 mmol/L</td>
<td>16–26 mmol/L</td>
</tr>
</tbody>
</table>

**Nursing Actions/Considerations**

- Place a jugular catheter.
- Initiate a fluid therapy plan.
- Monitor resolution of dehydration and restoration of intravascular volume.
- Monitor resolution of hyperglycemia and ketonuria.
- Monitor and document fluid “ins and outs.”
- Consider actions to be taken if pain, fluid overload, hypoglycemia, evidence of infection, hypo- or hyperkalemia, or catheter-related problems occur.

**Calculation of Lulu’s Fluid Therapy**

Replacement volume \((6 \text{ kg} \times 0.08)\) \(= 0.48 \text{ L}\)

Maintenance volume \((6 \text{ kg} \times 75 \text{ ml/kg/day})\) \(= 0.45 \text{ L}\)

Total volume \(= 0.93 \text{ L} \text{ or } 930 \text{ ml}\)

\[930 \text{ ml} \div 24 \text{ hours} = 39 \text{ ml/hour}\]

Note: Abnormal losses (i.e., vomiting, diarrhea, polyuria) should be made up by adding the previous hour’s abnormal losses to the next hour’s fluid input.

**Treatment and Response**

Initially, IM regular insulin therapy was initiated at 1 U/hour and then decreased to 0.5 U/hour. BG was measured hourly; fluids were supplemented with dextrose to
2.5%. When the BG level approached 250 mg/dl, the veterinarian was consulted (see Potential Adverse Conditions in Diabetic Ketoacidosis). The lactated Ringer’s solution was supplemented with 40 mEq/L potassium (split half with potassium chloride and half with potassium phosphate). IV piperacillin/tazobactam sodium (168 mg) was administered every 6 hours. IV famotidine (3 mg) was given every 24 hours. Serum electrolytes and venous blood gases were monitored every 4 hours. The poodle was given nothing by mouth. Temperature, pulse, and respiration were checked every 4 hours (for the first 24 hours) and then every 6 hours. IM oxymorphone (0.05 mg/kg) was administered every 4 hours as needed for pain.

Lulu’s glucose reached 220 mg/dl within 12 hours, and the ketonuria was resolved. With regard to the fluid therapy, 50% dextrose was added to make a 2.5% solution. The patient was started on NPH insulin. At the time of discharge (the sixth hospital day), Lulu was regulated on NPH insulin twice daily.

Summary

Diabetic ketoacidosis is a complex endocrine/metabolic disorder. After animals are diagnosed with DKA, a therapeutic plan should be implemented and the patient’s response to therapy monitored.

An IV catheter should be placed and fluids should be administered as directed by the veterinarian. Technicians must be capable of monitoring vital signs as well as the resolution of hypovolemia and dehydration and must also be able to recognize the signs of fluid overload and problems associated with catheterization. Insulin therapy must be properly administered. Technicians, therefore, must be aware of potential side effects as well as recognize the signs of insulin overdose.

Finally, technicians should be able to monitor the correction of acid-base and electrolyte abnormalities either through blood sample analysis or by assessing clinical signs and monitoring electrocardiographic readings. In summary, technicians must be able to recognize adverse situations as well as know how to treat them. A collaborative team effort gives patients the best chance of recovery.

References


About the Author

Mr. Davis is affiliated with the Emergency Nursing Service, Veterinary Medical Teaching Hospital, University of California, Davis, and is co-founder of the Academy of Veterinary Emergency and Critical Care Technicians. He has extensive experience in the field of emergency and critical care medicine.

The article you have read is the equivalent of ½ hour of study. To receive credit for your study, choose only the one best answer to each of the following questions; then mark your answers on the test form on the postage-paid envelope inserted in Veterinary Technician.

1. ___________ is not characteristic of DKA.
   a. Ketosis
   b. Alkalosis
   c. Dehydration
   d. Hyperglycemia
2. ___________ occurs when hyperglycemia exceeds the renal threshold.
   a. Potassium resorption
   b. Sodium resorption
   c. Osmotic diuresis
   d. Water retention

3. Which of the following is a potential risk factor for DKA?
   a. too little insulin
   b. increased insulin secretion
   c. increased ketone metabolism
   d. a high-fiber diet

4. ___________ has been reported to coexist with DKA.
   a. Respiratory distress syndrome
   b. A portosystemic shunt
   c. Glaucoma
   d. Cushing’s syndrome

5. Which of the following is not a goal of DKA therapy?
   a. reduction of total CO₂
   b. correction of dehydration
   c. normalization of BG level
   d. correction of serum electrolyte abnormalities

6. Which type of fluid places diabetic patients at risk for cerebral edema?
   a. plasma
   b. isotonic
   c. hypotonic
   d. whole blood

7. Insulin administration
   a. increases serum glucose.
   b. drives potassium into the cells.
   c. drives potassium out of the cells.
   d. increases ketone production.

8. Glucose reduction should not exceed ________ mg/dl/hour.
   a. 25
   b. 50
   c. 75
   d. 100

9. What is the likely cause if the following electrocardiographic changes occur: bradycardia, decreased P-wave amplitude, prolonged P-R interval and QRS complex, and spiked T wave?
   a. hypokalemia
   b. hyperkalemia
   c. hypercalcemia
   d. hypocalcemia

10. Which acid–base disturbance does NaHCO₃ correct?
    a. respiratory alkalosis
    b. respiratory acidosis
    c. metabolic acidosis
    d. metabolic alkalosis