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Diabetic Ketoacidosis: Treatment Recommendations*

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KEY FACTS

- Placement of a central sampling catheter and 24-hour nursing care can facilitate therapy and monitoring in diabetic ketoacidosis (DKA).
- Fluid therapy should be calculated for each patient and adjusted as hydration status changes.
- Hypokalemia, the most common electrolyte abnormality associated with DKA, may be exacerbated with fluid and insulin therapy.

ABSTRACT: Diabetic ketoacidosis (DKA) is a serious hormonal disorder requiring emergency therapy. Various potentially life-threatening fluid and electrolyte changes can occur with DKA. Development of a coordinated treatment plan to address fluid requirements, acid–base status, and multiple electrolyte abnormalities (e.g., hypokalemia, hypophosphatemia, hypomagnesemia) is imperative to successful therapy. Various insulin strategies may be used to control hyperglycemia and hyperketonemia.

Diabetic ketoacidosis (DKA) causes moderate to severe clinical illness. Diagnosis of DKA implies identification of hyperglycemia, glucosuria, ketonuria, and metabolic acidosis with concurrent clinical signs of dehydration, weakness, lethargy, vomiting, or anorexia. The initial therapeutic plan for patients with DKA should encompass all fluid, blood gas, and electrolyte disorders as well as reversing ketogenesis and resolving hyperglycemia. Development of a coordinated plan for fluid resuscitation, electrolyte supplementation, and initiation of insulin will increase the likelihood of successful therapy. Frequent patient monitoring and adjustment of therapy according to patient response will allow clinicians to tailor a plan to meet a patient's changing needs. This article outlines therapeutic recommendations for DKA, including electrolyte replacement and fluid, acid–base, and insulin therapies. Treatment recommendations are intended for patients with both biochemical and clinical signs consistent with moderate to severe DKA. Patients with diabetes mellitus (DM) and ketonuria that are eating and drinking without vomiting may be able to be managed more conservatively.

GENERAL THERAPEUTIC CONSIDERATIONS

Placement of an indwelling jugular or medial saphenous sampling catheter will facilitate intravenous (IV) fluid and drug therapy and permit blood sampling for necessary glucose, electrolyte, and blood gas evaluations.[1] Peripheral

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cephalic or medial saphenous catheterization will suffice for initial treatment and fluid resuscitation if placement of a jugular catheter is imprudent on an emergency basis. Patient safety should determine the method of catheter placement. Some therapies (e.g., IV regular insulin therapy) are best accomplished with two indwelling catheters: one catheter for blood sampling and fluid and/or drug administration and one for constant-rate infusion (CRI) of insulin.[1,2]

Many patients with DKA require therapy via CRI, and their clinical condition may deteriorate rapidly when hypoglycemia or electrolyte abnormalities occur suddenly. Patients that are seriously or critically ill require 24-hour supervision. If 24-hour monitoring is not available, conservative insulin therapy should be administered to avoid hypoglycemia, and the patient should be reassessed frequently by trained veterinary personnel.

INTRAVENOUS FLUID THERAPY

Fluid Selection

The goals of fluid therapy include volume resuscitation, rehydration, and correction of hyponatremia and hypochloremia. IV fluid therapy also lowers blood glucose by dilution and increases glomerular filtration rate to increase glucose loss.[3] Fluid therapy should be initiated with an isotonic sodium-based electrolyte solution (replacement crystalloid solution). Types of replacement crystalloid solutions include 0.9% sodium chloride (NaCl), lactated Ringer's solution, and Plasma-Lyte 148® (Baxter Healthcare Corporation, Deerfield, IL). Each of these fluids has a slightly different electrolyte composition. Plasma-Lyte 148® and lactated Ringer's solution contain bicarbonate precursors (i.e., acetate in Plasma-Lyte 148® and lactate in lactated Ringer's solution) that are metabolized to bicarbonate. Historically, the recommended replacement fluid for DKA treatment has been 0.9% NaCl.[1,4] Lactate must be metabolized in the liver in the same manner as ketone bodies, resulting in reduced hepatic lactate metabolism and elevation of circulating lactate levels. Because lactate is a negatively charged ion, further losses of sodium and potassium may occur with lactate excretion through the kidneys in an attempt to maintain electroneutrality.[1]

Hyponatremia

Hyponatremia is an anticipated finding with DM. Glucose plays an important role in increasing intravascular osmolality when circulating glucose levels are significantly elevated.[5] The following formula can be used to determine whether the degree of hyponatremia is appropriate for the degree of hyperglycemia[5]:

$$\text{Corrected } Na^+ = \text{Measured } Na^+ + \{1.6 \times [\text{Glucose (mg/dl)} - 100]/100\}$$

Sample Calculation of Initial Fluid Therapy

- **Patient:** 10% dehydrated 5-kg cat, vomits small amounts five times/day, normotensive
- **Maintenance needs:** 60 ml/kg/day x 5 kg = 300 ml/day
- **Volume to correct dehydration:** 5 kg × 0.10 × 1000 ml/kg = 500 ml
- **Ongoing losses:** 25 ml/day
- **Total fluid requirement:** 825 ml
- **Amount of fluid administered by CRI over 24 hr:** 34 ml/hr

If the corrected sodium is in the normal range, hyponatremia is due to free water gain in the intravascular space and measured sodium should normalize with reduction of blood glucose. If the corrected sodium is still below normal, sodium wasting has also occurred from chronic diuresis and 0.9% NaCl is the fluid treatment of choice to replace the sodium deficit.

Normal serum sodium in the face of hyperglycemia is inappropriate and represents excess free water loss and hyperosmolality. An isotonic replacement crystalloid solution is still indicated until dehydration has been corrected.[1] Hypotonic fluids (e.g., 0.45% NaCl) should be avoided initially because rapid shifts in osmolality can precipitate cerebral edema and coma in DKA patients. Fluids such as 0.45% NaCl with 2.5% dextrose are isotonic at the time of administration but become hypotonic shortly thereafter as glucose is metabolized.[6]

Quantity and Rate of Administration

Fluid dose should be calculated to include maintenance fluid requirements, dehydration, and contemporary losses from vomiting or diarrhea (see Calculation of Initial Fluid Therapy). Estimating fluid dose without calculation may cause the fluid requirement to be underestimated, especially if marked dehydration is present. Maintenance fluid requirement can be calculated at a dose of 55 to 65 ml/kg/day.[6] This requirement combines insensible losses (e.g., water used for metabolic reactions, normal fecal losses, ventilation, mucous membrane evaporation) of 20 ml/kg/day and estimated urine losses of 35 to 45 ml/kg/day. Maintenance needs should be adjusted daily based on the patient's response and hydration status. The maintenance fluid dose should be given over 24 hours.[6]

Volume of fluid needed to correct dehydration is based on the estimate of the percent of dehydration from clinical parameters. The following formula is used to translate calculated dehydration into fluid requirement[1]:

Figure 1A

Figure 1B

Figure 1–Clinical signs of hypokalemia include cervical ventroflexion (**A**) and severe muscle weakness (**B**).

Dehydration dose = Estimated dehydration (0.00) × Body weight in kg × 1000 ml/kg

Rate of administration of the dehydration dose is most often divided over 12 to 24 hours and added to the maintenance fluid dose. In hypovolemic or hypotensive patients, a fluid dose to approximate one blood volume (60 ml/kg for cats, 80 ml/kg for dogs) may be given within the first 1 to 2 hours of treatment to expand the vascular space. Patients should be reassessed to determine their response to treatment before the remaining daily fluid dose is administered.[6] Excessively rapid volume administration may result in hypervolemia and may cause fluid precipitation into the extravascular space, possibly leading to pulmonary edema.[6] If a central venous catheter is appropriately positioned, central venous pressure can be monitored to help avoid excessive vascular expansion. Contemporary losses from vomiting or diarrhea should be estimated and added to the calculated fluid requirement. This figure can be altered daily if vomiting or diarrhea resolves.[6]

Fluid should be administered by a CRI pump, if available. Alternatively, careful manual adjustment of rate and visual observation of fluid volume may be used; however, this method is not recommended if potassium requirements are high or other additives (phosphorus or magnesium) are prescribed.[1] Timely evaluation of hydration status is imperative to avoid overhydration, especially with a large volume of fluid administration. Hydration may be estimated by skin turgor and mucous membrane feel and more objectively by monitoring body weight at least twice daily. Fluid dose should be recalculated and tailored to meet each patient's needs.[6]

All replacement crystalloid solutions are potassium-poor and may exacerbate hypokalemia and hypophosphatemia. Hyperglycemia will be positively affected by dilution with fluid administration.

TABLE 1
Potassium Replacement Therapy for Patients with Diabetic Ketoacidosis[a]

Serum Potassium Level (mEq/L)	*Potassium Supplementation Required (mEq)/L*
3.5–5	20
3.0–3.4	30
2.5–2.9	40
2.0–2.4	60
<2.0	80

[a]Therapy must not exceed 0.5 mEq/kg/hr.

POTASSIUM THERAPY

Hypokalemia is the most common electrolyte abnormality associated with DKA.[1,7–9] Clinical signs most commonly associated with hypokalemia include muscle weakness, cervical ventroflexion in cats, cardiac arrhythmia, and respiratory muscle failure in severely affected patients (Figure 1).[6,10] Potassium therapy in emergency settings consists of IV therapy via CRI using one of two dosing methods, based on the patient's measured plasma or serum potassium level. Blood potassium should be monitored at regular intervals to avoid iatrogenic hyperkalemia.

Mild to moderate hypokalemia—Potassium chloride (KCl) should be added to the IV fluids to correspond with the patient's serum potassium level. The amount to be added should be based on a potassium replacement scale (Table 1).[11] Possible shortcomings of this scale include underestimation of patient potassium needs with small to moderate fluid volume or overdosage of KCl with a high rate of fluid administration for animals with severe dehydration.

Moderate to severe hypokalemia—Potassium replacement should be calculated rather than estimated to avoid potential underdosage or overdosage of KCl (see Sample Potassium Replacement Calculation, p. 333). Traditional guidelines recommend administration at a rate of less than 0.5 mEq/kg/hour.[12] When administering KCl to animals with severe hypokalemia, potassium levels should be rechecked within 6 to 8 hours to

Potassium Replacement Calculation

- **Patient:** 5% dehydrated 5-kg cat with severe hypokalemia
- **Calculated fluid requirement:** 23 ml/hr
- **Potassium level:** 1.7 mEq/L
- **Calculated potassium replacement:** 0.5 mEq/kg/hr × 5 kg = 2.5 mEq/hr
- **Amount of potassium chloride (2 mEqK^+/ml) in 1 L fluid:** 109 mEq/L administered at 23 ml/hr
- **Estimated potassium chloride dose using replacement scale from Table 1:** 80 mEq/L

allow adjustment of therapy as needed. In humans with life-threatening hypokalemia (serum potassium less than 2 mEq/L, showing signs of severe weakness), potassium may be given at a dose that approximates 0.5 to 1.0 mEq/kg/hour for the first hour of therapy followed by a reassessment.[13] Withholding insulin and bicarbonate therapy and avoiding rapid administration of IV fluids until potassium therapy has been initiated may help avoid iatrogenic exacerbation of hypokalemia in these patients.[13]

PHOSPHORUS THERAPY

Hypophosphatemia may be recognized initially or following DKA treatment. Patients with biochemical evidence of hypophosphatemia or asymptomatic patients deemed to be at risk of developing hypophosphatemia may be treated by adding potassium phosphate (KPO_4) to IV fluids via CRI.[14,15] In the literature, the initial dose of KPO_4 is given via CRI at 0.01 to 0.03 mmol/kg/hour with 0.9% NaCl for the first 6 hours. Serum phosphorus level should then be reevaluated and the dose adjusted if necessary.[10,15] In severely phosphorus-depleted animals, the dose may need to be increased to 0.03 to 0.12 mmol/kg/hour.[16,17] Alternatively, phosphorus may be supplemented by administering half of the potassium requirement as KPO_4.[3] However, this protocol can result in phosphorus overdose when the potassium deficit exceeds the phosphorus deficit.[18]

Higher phosphorus doses necessitate increased frequency of serum phosphorus monitoring. Hyperphosphatemia should be avoided to prevent mineralization of tissues and acute tetany from secondary hypocalcemia.[10] Monitoring serum calcium levels during therapy is prudent to identify iatrogenic hypocalcemia before the onset of clinical signs.[14] Packed cell volume should be measured at the outset of therapy and monitored frequently because hypophosphatemia may result in acute-onset hemolytic anemia.[16] Transfusion therapy may be indicated if anemia is severe.[16]

ACID–BASE THERAPY

High anion gap metabolic acidosis is common in DKA, both from ketoacidosis and from lactic acidosis secondary to dehydration.[6] Clinical manifestations of metabolic acidosis include lethargy, vomiting, hyperventilation, decreased myocardial contractility, peripheral vasodilation, stupor, and coma.[6] Administration of insulin to reverse ketogenesis and IV fluid therapy to correct dehydration will result in improvement of metabolic acidosis in most patients.[19,20]

Bicarbonate administration to correct metabolic acidosis in patients with DKA remains controversial. In humans, no evidence has shown that survival improves or clinical outcome changes with bicarbonate therapy.[21,22] In addition, aggressive bicarbonate therapy can lead to paradoxical central nervous system acidosis, coma, and death.[17,20,21] If the patient is alert and mentally stable, bicarbonate therapy should be withheld.[3] In patients with severe acidemia (blood pH level less than 7.10 to 7.15, bicarbonate values less than 8 mmol/L, and clinical signs referable to metabolic acidosis), bicarbonate therapy can be calculated using the following formula[6]:

$$\text{Bicarbonate deficit} = \text{Base deficit} \times \text{Body weight in kg} \times 0.3$$

Administer half of the calculated deficit in IV fluids over 4 to 6 hours, and then reassess blood gas to determine whether further therapy is needed.[6]

MAGNESIUM THERAPY

Hypomagnesemia has become a more frequently recognized complication of DKA.[23,24] Hypomagnesemia may be mild and clinically inapparent or may be severe. Mild hypomagnesemia may be treated by administering isotonic replacement crystalloid solutions that contain magnesium (Plasma-Lyte 148®).[10] For animals with severe hypomagnesemia (serum magnesium less than 1.2 mg/dl), an infusion of magnesium sulfate can be made by adding magnesium sulfate to 5% dextrose in water; for the first 24 hours, an initial dose of 0.75 to 1 mEq/kg/day CRI can be administered. The dose should then be reduced by 50% for the next 3 to 5 days.[10,23,25]

INSULIN THERAPY

General Considerations

Insulin reduces blood glucose by promoting cellular glucose uptake and decreasing hepatic glucose production.[4] Ketogenesis is reversed by reducing available substrate for ketone formation, limiting hepatic production by reducing glucagon levels, and increasing peripheral ketone use.[4] In addition to these beneficial effects, insulin therapy can contribute to life-threaten-

TABLE 2
Insulin Adjustment with Changes in Blood Glucose Concentration in Dogs with Diabetic Ketoacidosis[a]

Blood Glucose Concentration (mg/dl)	*Solution to Fulfill Fluid Requirements*	*Rate of Administration of Insulin Solution (ml/hr)[b]*
>250	0.9% NaCl	10
200–250	0.45% NaCl + 2.5% dextrose	7
150–200	0.45% NaCl + 2.5% dextrose	5
100–150	0.45% NaCl + 2.5% dextrose	5
<100	0.45% NaCl + 5% dextrose	Stop insulin infusion

[a]From Macintire DK: Treatment of diabetic ketoacidosis in dogs by continuous low-dose intravenous infusion of insulin. *JAVMA* 202(8):1271, 1993; with permission.
[b]Solution comprised of regular crystalline insulin at a dose of 2.2 U/kg/day for dogs (1.1 U/kg/day for cats) added to 250 ml of 0.9% NaCl or Ringer's solution.
NaCl = sodium chloride.

ing complications such as hypoglycemia, hypokalemia, and sudden changes in osmolality.[26] Appropriate therapy tailored to individual patients and consistent patient monitoring can help avoid these serious complications.

Selection of Insulin

Only regular insulin should be used in the initial treatment of DKA.[1,26,27] Regular insulin has a rapid onset of activity and short duration of action.[26,28] In addition, regular insulin can be administered IV, intramuscularly (IM), or subcutaneously (SQ). When the patient is stabilized, insulin with a longer duration of activity is indicated for long-term maintenance.

Insulin Protocols

Three insulin protocols for initial management of DKA have been described in the veterinary literature: IV CRI, hourly IM insulin, and IM insulin administered every 4 to 6 hours.[9,26,29] All three protocols can reduce blood glucose and reverse ketogenesis. Because each protocol has advantages and disadvantages, the experience and preference of the managing clinician should determine protocol selection. Similarities between the three protocols include the use of regular insulin to gradually lower blood glucose, administration of 2.5% to 5% dextrose in IV fluids when blood glucose falls below 250 mg/dl, frequent assessment of individual patient response to therapy, and change to intermediate-acting insulin for long-term outpatient management once ketogenesis has resolved and the clinical condition improved.

Intravenous Constant-Rate Infusion

Intravenous CRI is the treatment of choice for human patients in DKA crisis in intensive care units. This method is also safe and effective for controlling DKA in veterinary patients.[4,5,9,27] A starting dose of 2.2 U/kg/day regular insulin for dogs or 1.1 U/kg/day for cats is added to 0.9% NaCl and administered by CRI; the rate of insulin-containing fluid is adjusted depending on the rate of change of patient blood glucose (Table 2).[1] This initial infusion rate provides a dose of 0.09 U/kg/hour for dogs and 0.045 U/kg/hour for cats.[9] The lower dose is recommended for cats to reduce sudden osmolar shifts that might lead to cerebral edema.[1] Because insulin binds to plastic and glass, 50 ml of insulin-containing fluid solution should be allowed to run through the IV line before administration.[1]

Two separate catheters are recommended for treatment: a peripheral catheter for administration of insulin solution and a central sampling catheter for administration of any fluid needed in addition to that administered with insulin so that fluid adjustments may be made easily based on patient needs.[27] Alternatively, insulin solution can be piggy-backed onto an existing fluid line if a separate infusion pump is used.[1] Blood glucose should be monitored at 1- to 2-hour intervals. Insulin administration rate should be increased or decreased to reduce blood glucose levels by a rate of 50 to 70 mg/dl/hour.[1] Rate of administration of insulin-containing fluid should be adjusted based on serial blood glucose measurements to insure that blood glucose levels remain above 250 mg/dl for the first 4 to 6 hours of treatment to avoid risk of cerebral edema from rapid osmolar shifts.[1] When blood glucose levels reach 200 to 250 mg/dl, 2.5% to 5% dextrose solution should be administered with the IV fluids to avoid hypoglycemia while continuing to promote ketone metabolism through insulin administration (Table 2).[1] When ketosis has resolved and the patient is eating and drinking without vomiting, treatment can be switched to twice-daily insulin therapy with an intermediate-acting insulin such as NPH or Lente insulin.[1]

This protocol can safely and effectively reduce blood

TABLE 3
Rate of Insulin and Fluid Administration for a 10-kg Dog Based on Four-Times-Daily Blood Glucose Determination[a]

Blood Glucose Level (mg/dl)	*Fluid Therapy*	*Regular Insulin Dose*
>500	0.9% NaCl	4
400–500	0.9% NaCl	3
250–400	0.9% NaCl	2
200–250	0.45% NaCl + 2.5% dextrose	2
100–200	0.45% NaCl + 2.5% dextrose	1
60–100	0.45% NaCl + 2.5% dextrose	No insulin
<60	0.45% NaCl + 5% dextrose	No insulin

[a]Insulin doses can be altered depending on individual response to therapy over time.
NaCl = sodium chloride.

glucose and reverse ketogenesis. It is less likely to stimulate glucagon and cortisol release than are intermittent injections.[9] Disadvantages include the need for more intensive nursing care with frequent glucose monitoring and the requirement for CRI administration.[26] The risk of excessively rapid reduction of blood glucose and electrolytes exists if the patient is not monitored closely.[26]

Hourly Intramuscular Insulin

Hourly IM insulin protocols have been described for veterinary patients.[3,4,29] For the hourly protocol, regular insulin should be administered at an initial dose of 0.2 U/kg IM, then 0.1 U/kg hourly thereafter.[19] For dogs and cats requiring very small doses of insulin, use of 0.3-ml, U-100 insulin syringes may facilitate appropriate dosing.[30] Regular U-100 insulin may be diluted 1:10 with the appropriate pH-adjusted diluent available from the manufacturer for the specific insulin being used.[30] Isotonic saline may also be used as a diluent when manufacturer diluents are unavailable; however, the mixture may not have a shelf life beyond 24 hours.[31] Blood glucose should be monitored hourly

with the sodium; a gradual decline of 50 to 100 mg/dl/hour is needed to avoid sudden osmolar shifts.[3] When blood glucose concentration falls below 300 mg/dl, 5% dextrose solution should be added to the IV fluids and dosing frequency should be reduced to 4- to 6-hour intervals IM or SQ to keep blood glucose between 200 to 300 mg/dl.[3] Once the patient is eating and drinking without vomiting, the protocol should be changed to a twice-daily SQ intermediate-acting insulin.[3]

Similar to the IV CRI protocol, the hourly protocol can be effective with appropriate use and monitoring. This protocol has the disadvantage of requiring hourly blood glucose measurements and intensive management by trained professional staff. Care must be taken to insure deep IM injection in the dehydrated patient to avoid erratic absorption from SQ administration.

Intramuscular Intermittent Insulin

The IM intermittent protocol is practical, safe, and reliable and may be better suited to a private practice setting because less frequent blood glucose sampling and treatment changes are required.[26] For this protocol, 0.25 U/kg regular insulin should be administered IM as a test dose, with subsequent doses based on the patient's response to initial treatment.[26] In obese animals, initial dose should be based on estimated lean body weight to avoid overdosage and hypoglycemia. Blood glucose should be rechecked at 4- to 6-hour intervals; the goal is to reduce blood glucose by 50 to 70 mg/dl/hour.[26] If this goal is surpassed, the next insulin dose should be reduced by 25% to 50%. If this goal is not met, the next dose should be increased by 25% to 50%. If the blood glucose level reaches 200 to 250 mg/dl, 2.5% to 5% dextrose should be added to the IV fluids.[26] When the patient's general response to insulin is known, a chart may be provided to direct the veterinary technician to administer subsequent insulin doses based on blood glucose levels (Table 3).[26] Similar to other protocols, intermediate-acting insulin should be administered SQ twice daily when ketosis has resolved and the patient is eating and drinking without vomiting.[26]

Advantages of the IM intermittent protocol include treatment via one IV catheter, less frequent sampling times, gentle reduction of blood glucose level, and resolution of ketogenesis.[26] One possible disadvantage is that ketogenesis may take longer to resolve with intermittent insulin injections because regular insulin has a variable duration of activity and may not be present for the full 6 hours between administration.[9] Additionally, if the duration of insulin is shorter than 6 hours in an individual patient, measurement of blood glucose at a 6-hour interval may not accurately reflect glycemic control. In cases in which blood glucose concentrations

are difficult to control, more frequent monitoring may be indicated. Controlled, prospective studies comparing clinical response between all protocols are lacking in veterinary patients.

PATIENT MONITORING

In addition to blood glucose monitoring as recommended by the selected insulin protocol, blood gas and electrolyte monitoring should be continued on a regular basis according to the clinician's discretion and based on the clinical status of the patient. Electrolyte abnormalities may need to be monitored as frequently as two to four times a day on the first day and less frequently when the patient is responding to therapy. Electrolyte changes should be addressed as needed by altering fluid composition. Packed cell volume and plasma hemolysis should be monitored in hypophosphatemic patients to evaluate for acute hemolytic anemia.[16]

As ketogenesis is reversed, the anion gap will return to normal and urine ketones will resolve. Serum β-hydroxybutyric acid is metabolized to acetoacetic acid before being eliminated. Because urine dipsticks measure acetoacetic acid, urine ketones may increase temporarily while serum ketones are being metabolized.[3]

Monitoring hydration status and adjusting fluid rate accordingly are imperative given the high initial fluid rates required in DKA patients. As hyperglycemia and ketoacidosis resolve, osmotic diuresis and urine fluid losses should also resolve. Overhydration, with subsequent pulmonary edema or pleural effusion, may result if fluid continues to be given at high rates to the hydrated, volume-expanded patient.[6]

PROGNOSIS

Diabetic ketoacidosis remains a serious metabolic illness with a guarded prognosis for both human and veterinary patients.[1,3,32] In two retrospective studies outlining response to treatment for dogs and cats diagnosed with DM or DKA, 29% of the dogs and 26% of the cats died or were euthanized following initial diagnosis.[9,33] Patients with DKA frequently have concurrent disease processes (e.g., pancreatitis, renal disease) that may decrease the chances of successful therapy.[7–9,33] Some clients may elect euthanasia at the outset because of the serious nature of DKA and long-term care required for DM.[3,8]

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ARTICLE #3 CE TEST

The article you have read qualifies for 1.5 contact hours of Continuing Education Credit from the Auburn University College of Veterinary Medicine. *Choose the one best answer* to each of the following questions; then mark your answers on the test form inserted in *Compendium*.

1. _____ is not recommended for managing patients with DKA.
   a. Placement of a peripheral catheter
   b. Placement of a central sampling catheter
   c. Twenty-four–hour nursing care
   d. Placement of an indwelling urinary catheter
   e. Monitoring fluid infusion

2. Normal serum sodium in a hyperglycemic patient with DM
   a. indicates normal serum osmolality.
   b. requires that fluid therapy with 0.45% NaCl be instituted.
   c. is an indication of free water loss.
   d. is considered appropriate.
   e. should be followed by fluid therapy with 0.9% NaCl to exacerbate free water loss.

3. When prescribing fluid therapy for patients with DKA,
   a. the dehydration component should replace losses from vomiting and diarrhea.
   b. the maintenance fluid requirement should be given over 12 hours when dehydration is present.
   c. the requirements can be estimated quickly by doubling the maintenance dose.
   d. a CRI pump is a requirement.
   e. body weight, skin turgor, and mucous membranes should be assessed frequently.

4. Which of the following mechanisms does not contribute to hypokalemia in patients with DKA?
   a. intracellular shifting from chronic metabolic acidosis
   b. poor potassium intake from loss of appetite
   c. hyperglycemia causing osmotic diuresis and renal potassium loss
   d. insulin therapy driving potassium intracellularly
   e. vomiting causing ongoing potassium loss

5. _____ is not a treatment consideration for patients with hypophosphatemia.
   a. Administering therapy via CRI pump
   b. Administering KPO_4 at a dose range of 0.03 to 0.12 mmol/kg/hour
   c. Administering therapy for 48 hours (then rechecking phosphorus)
   d. Monitoring packed cell volume

6. Bicarbonate
   a. should be administered to DKA patients with pH levels below 7.35.
   b. should be administered to all patients with DKA.
   c. administration provides excellent clinical benefits to most DKA patients.
   d. should be given only to patients with clinical signs of metabolic acidosis.
   e. administration will only correct ketoacidosis, not lactic acidosis.

7. Serum total magnesium
   a. is commonly low on presentation in patients with DKA.
   b. provides excellent correlation with total body magnesium.
   c. generally becomes low quickly with low dietary intake.
   d. represents more than 1% of total body magnesium.
   e. should be measured following fluid treatment in patients at risk of hypomagnesemia.

8. Which of the following statements regarding insulin therapy for patients with DKA is true?
   a. NPH or Lente insulin is the first choice for initial treatment.
   b. SQ administration is usually appropriate for initial treatment.
   c. IV CRI insulin therapy effectively reverses ketogenesis.
   d. Only high doses of insulin will reverse ketogenesis.
   e. IV dextrose is contraindicated with hourly IM insulin.

9. Which of the following statements regarding DKA protocols is true?
   a. Intermediate-acting insulin is appropriate when ketosis is resolved.
   b. Intermediate-acting insulin is appropriate when blood glucose is less than 200 mg/dl.
   c. Regular insulin should be used until patients are discharged.
   d. Blood glucose should decline no more rapidly than 25 mg/dl/hour.
   e. IV dextrose should be initiated when blood glucose falls below 400 mg/dl.

10. In general, patients with DKA have
    a. an excellent prognosis for long-term recovery.
    b. a good prognosis if hypokalemia is not present.
    c. a poor prognosis because many animals are euthanized before treatment is initiated.
    d. a guarded prognosis because of serious metabolic changes.