# Recognition, diagnosis and care of the diabetic ketoacidotic patient

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**ABSTRACT** Diabetic ketoacidosis is a potentially fatal condition if not recognised and acted on swiftly. However, following prompt diagnosis and treatment from the veterinary surgeon and with intensive care from the nursing team, these patients can often make a good recovery. This article discusses the clinical signs, diagnostic aids and care required for the treatment of this patient.

**Keywords** diabetes mellitus, diabetic ketoacidosis, nursing care, diagnosis

## Introduction

Ketoacidosis is the excessive production of ketoacids circulating in the blood, leading to metabolic acidosis and acidaemia. Small amounts of ketoacids can be found in healthy animals but this is not usually enough to cause a disturbance. Ketoacidosis is invariably a serious and life-threatening complication of diabetes mellitus (DM), which is often picked up at the time of diagnosis but can also occur in diabetic patients already on treatment.

During diabetic ketoacidosis (DKA) the body begins to produce ketone bodies as an alternative energy source from free fatty acids in the liver (Gear & Mathie, 2011). Counter-regulatory hormones such as glucagon, cortisol, growth hormone and catecholamines dominate over a reduced or negligible insulin output, which most commonly occurs when there is a secondary triggering disease, as shown in **Table 1** (Skelly, 2018).

The lack of insulin allows free fatty acids to be released into the circulation and taken up by the liver for ketoacid production (Skelly, 2018). While ketone bodies can be adequately used as a source of energy, animals in DKA produce ketones that exceed the rate of utilisation, resulting in ketonaemia, ketonuria and acidosis.

**Table 1.** Conditions that may trigger diabetic ketoacidosis (Boag, 2012).

| | |
|---|---|
| **Bacterial infections** | Urinary tract infection<br>Pneumonia<br>Pyometra<br>Pyoderma<br>Prostatitis |
| **Inflammatory disease** | Pancreatitis |
| **Endocrinopathy** | Hyperadrenocorticism<br>Hypothyroidism (dog)<br>Hyperthyroidism (cat)<br>Acromegaly |
| **Other** | Chronic renal failure<br>Neoplasia<br>Corticosteroid administration |

## Clinical history

Most cases of DKA will have a history of polyuria, polydipsia, weight loss, muscle wastage and deeper, faster respiration reflecting metabolic acidosis (Battaglia & Steele, 2016; Skelly, 2018). On presentation there will be weakness, depression to stupor, tachypnoea, anorexia, vomiting and hepatomegaly (Gear et al., 2011; Battaglia & Steele, 2016; Skelly, 2018). In some circumstances, the patient may present with ketotic halitosis, although it is said not everyone can recognise the smell so this should not be considered a definitive symptom (VetsNow, 2013). Should the patient already have been diagnosed with DM, the owner should be questioned about any changes in insulin administration (Boag, 2012).

## Diagnostics

Various diagnostics can be performed to aid in the diagnosis of DKA. Skelly (2018) state the tests seen in **Table 2** can be used in most collapsed and volume-depleted patients to aid faster diagnosis. This table shows the recommended diagnostics and a brief overview of expected findings.

In particular, with a patient that is suspected to be in DKA, the VS should ask for a dipstick analysis of the patient's urine to assess for ketonuria.

**Table 2.** Diagnostic tests used to detect diabetic ketoacidosis. PCV, packed cell volume.

| Diagnostic test | Expected findings |
|---|---|
| **PCV/total solids** | Increased due to dehydration |
| **Blood glucose** | Increased due to unstable DM |
| **Dipstick analysis** | Ketones and glucose |
| **Urine culture analysis** | Urine culture may show signs of underlying urine infection as a triggering disease |
| **Blood electrolytes** | Sodium and chloride may vary from low to normal or high reflecting the balance of free water vs electrolyte loss. Hypokalaemia or hyperkalaemia can be expected, with hypokalaemia being more common |
| **Renal function assessment** | May present as azotaemic |
| **Blood gas analysis** | Low pH and negative base excess |

This can be performed by the dedicated laboratory technician (Skelly, 2018). A free-flow urine sample can be collected and the test performed in-house, with a result available within minutes.

The three main ketone bodies found in the urine are beta-hydroxybutyrate, acetate and acetoacetate. These are produced by the liver and used as an energy source when glucose is not readily available. Commercial dipsticks are available for the testing of ketones in the urine, but they only detect the presence of acetate and acetoacetate, not beta-hydroxybutyrate (VetsNow, 2013).

Boag (2012) states that beta-hydroxybutyrate is produced in larger quantities than the other acids when the body is in a state of hyperfusion and shock, so the presence of this acid will allow a more clear diagnosis of DKA. If the ketones present are beta-hydroxybutyrate but are not detected, it is possible to dilute the urine 1:9 with hydrogen peroxide to turn the beta-hydroxybutyrate to acetoacetate to make the ketones detectable (VetsNow, 2013).

Urinalysis is a crucial part of the diagnosis and should include specific gravity, sediment examination and general dipstick evaluation, as well as a ketone-specific dipstick. A urine culture is generally advised to test for an underlying urinary tract infection and is considered essential if the sediment examination is suggestive of infection. Diabetic patients are particularly prone to urinary tract infections, which can lead to destabilisation of the diabetes resulting in DKA (VetsNow, 2013). It is crucial, during the diagnostics stage, to try to identify the underlying cause of the condition, which will need to be corrected alongside the acidosis.

DKA patients commonly present with azotaemia. This is often prerenal secondary to dehydration. The nature of the azotaemia (renal or prerenal) can be assessed by performing a specific gravity (SG) measurement on the urine. However, due to diabetes leading to osmotic diuresis enhanced by ketonuria, the urine SG results cannot be completely relied on to be precise. Due to the condition and complicating factors, the patient will commonly have a urine SG of >1.020 (Boag, 2012).

A blood gas analysis should be performed to allow quantification of the blood acidity (Boag & Nichols, 2011). A blood gas analyser is used to measure blood pH, partial pressure of oxygen, partial pressure of carbon dioxide, bicarbonate levels, base excess and saturation of haemoglobin with oxygen. These parameters are used to indicate metabolic disturbances (Boag & Nichols, 2011). Initial and continuous blood glucose measurements will need to be taken throughout the treatment process. The normal blood glucose range is 3.3–6 mmol/l (Irwin-Porter, 2011). A patient in DKA will have a blood glucose higher than the normal range (Gear & Mathie, 2011).

In both cats and dogs, hyperglycaemia is often accompanied by stress. This may lead to confusion in the diagnosis of DM. When assessing blood glucose, it is beneficial to take a series of samples once the patient is settled to allow complete monitoring. In a routine blood-glucose curve these samples would usually be taken over 8–12 hours, at 2-hourly intervals, but the more critical patient would benefit from more regular sampling. Skelly (2018) states that there is no cut-off value to distinguish between stress and true DM, but it is rare for stress alone to cause a blood glucose of >20 mmol/l.

The blood samples can be taken via ear prick or venous sampling. An ear prick would be perfectly acceptable for a one-off sample. However, for continuity, it would be advisable to take venous samples, as ear-prick samples can differ depending on the patient's tissue perfusion. When necessary, venous sampling can be performed with a needle and syringe or, for better patient care, a central line can be placed. This would allow for continuous blood sampling without the need to frequently insert a needle into the patient.

## Care of the patient

Intravenous fluid therapy is required in all DKA patients, due to the dehydration that comes secondary to diuresis from hyperglycaemia (Battaglia & Steele, 2016). If the patient arrives in shock, over 48 hours of aggressive fluid therapy should be given to replace deficits in addition to maintenance fluids and ongoing losses (Battaglia & Steele, 2016). The fluid supplementation given over 24 hours can be calculated based on:

**Deficit (ml) =**
**percentage dehydration (%) × weight (kg) × 10**

**+**

**Maintenance (ml) = 50 ml × weight (kg)**

**+**

**Ongoing losses**

Ongoing losses will be individual to each patient depending on ongoing gastrointestinal losses and renal losses from osmotic diuresis (Boag, 2012). Hypovolaemia severity can be assessed by different clinical signs, which can be seen in **Table 3**.

When using fluid therapy to assist in the management of DKA, the correction of the acidosis and dehydration is the primary concern, alongside the patient being on insulin therapy (Skelly, 2018). The choice of fluid therapy is dependent on the hydration status and electrolyte status of the patient, but there are no published studies comparing the efficacy of different fluid types in critically ill diabetic patients. Although some clinicians recommend 0.9% sodium chloride,

**Table 3.** Stages of hypovolaemia and associated clinical signs (Boag, 2012). CRT, Capillary refill time; MM = mucous membranes.

| Clinical parameter | Mild hypovolaemia | Moderate hypovolaemia | Severe hypovolaemia |
|---|---|---|---|
| **Heart rate** | 130–150 bpm | 150–170 bpm | 170–220 bpm |
| **MM colour** | Normal | Pale pink | Grey/muddy |
| **CRT** | Rapid <1 second | 1–2 seconds | >2 seconds or absent |
| **Pulse amplitude** | Increased | Mild to moderately decreased | Severely decreased |
| **Pulse duration** | Mildly reduced | Moderately reduced | Severely reduced |
| **Metatarsal pulse** | Easily palpable | Just palpable | Absent |

many clinicians avoid acidic unbuffered fluids, opting instead for a balanced buffered isotonic solution – such as Plasma-Lyte or lactated ringer's solution (Hartmann's) – to limit the potential for the development of hyperchloraemic metabolic acidosis (Skelly, 2018). Boag (2012) recommends that the fluid type chosen should have a sodium concentration close to the patient's serum sodium, to avoid rapid changes in their serum sodium concentration.

Should the patient present with severe hyponatraemia, and is started on 0.9% sodium chloride, the patient should be transitioned to a more balanced solution after 24 hours of therapy (Battaglia & Steele, 2016). Lactated ringer's solution may also be more beneficial in patients where the blood pH is extremely acidic, as 0.9% saline can contribute to acidosis through a dilution effect (Battaglia & Steele, 2016; Skelly, 2018). A general disadvantage of long-term use of isotonic fluid therapy is the increased chance of hypokalaemia. However, due to the condition, this will most likely be an abnormality in need of correction anyway (Aldridge & O'Dwyer, 2013).

It is recommended to begin insulin treatment 4–6 hours after starting fluid therapy, to ensure the hypovolaemia is being corrected (Battaglia & Steele, 2016). The clinical symptoms of stages of hypovolaemia can be seen in **Table 3**. Insulin therapy is the cornerstone of treatment, as it allows glucose to be taken up by cells for metabolism and prevents further lipolysis from adding to the ketone burden and promotes ketone metabolism (Skelly, 2018).

There are three types of insulin that can be administered to a diabetic patient: soluble (neutral) insulin (short-term use), lente insulin (medium-acting use) and protamine zinc insulin (long-acting, most commonly used on stable long-term diabetic patients). There are two methods of insulin treatment in a DKA patient: intravenous constant-rate infusion (CRI) and short-acting single injections. Short-acting injections can be given subcutaneously or intramuscularly for quicker treatment reactions.

A CRI is the constant delivery of any intravenous medication that can be adjusted whenever necessary, according to clinical symptoms (Gear & Mathie 2011). Insulin CRIs are administered using a neutral insulin to allow for an instant change of rate if necessary. This can be given at a rate of 0.05–0.5 IU/kg/hour (Battaglia & Steele, 2016). A CRI is appropriate in most cases of DKA but is predominantly required in moderate to severe cases. With a patient that is showing mild clinical signs, single injections may be sufficient to manage the case (Skelly, 2018).

For accuracy, the insulin CRI should be given using a separate fluid bag and attached to a drip pump, to allow for changes of the CRI rate without having to change the rate of fluids being used for rehydration.

It should be noted that, prior to administration, 50 ml of the insulin fluid solution should be run through the giving set, as insulin binds to the tubing and the syringe. The solution creates a coating that facilitates successful administration (VetsNow, 2013).

The use of a CRI requires at least one fluid pump, so the choice to use this method will need to be in accordance with equipment availability, depending on the resources of the clinic (Battaglia & Steele, 2016).

Another insulin protocol involves injecting the patient intramuscularly with neutral insulin (Skelly, 2018). The initial dose is given at 0.2 IU/kg and, following hourly blood-glucose measurements, further 0.1 IU/kg doses are administered until blood glucose normalises (VetsNow, 2013). Once the blood glucose has normalised and the patient no longer shows signs of ketosis, longer-acting insulin can be administered subcutaneously (VetsNow, 2013).

Depending on the stage of ketosis, this method should be carefully considered as it involves the repeated injection of the patient, alongside repeated blood taking for glucose measurements. It should also be considered that giving any injection intramuscularly has a delayed acting time of 10–30 minutes (Battaglia & Steele, 2016).

When beginning insulin therapy, care must be taken not to reduce the glucose levels too rapidly. The serum glucose concentration decline should be 2.7–5.5 mmol/l/hour. Should the levels decline more rapidly, significant complications can occur (Battaglia & Steele, 2016). The brain produces small molecules that attract water, which maintains hydration of the brain when it is a hyperosmolar environment. These osmoles are not instantly removed when treatment has started, so rapid rehydration of the patient can occur, causing cerebral oedema, seizures, coma and death (Battaglia & Steele, 2016).

## Additional considerations

During DKA, the potassium will shift out of the cells into the serum to attempt to replenish renal losses and offset acid–base imbalances (Battaglia & Steele, 2016). As a result, the patient will have a total-body depletion of potassium and will require potassium supplementation on arrival and throughout the treatment (**Table 4**). During the treatment of DKA, potassium levels will continue to deplete due to dilution from aggressive fluid therapy, insulin-mediated uptake of potassium by the cells, correction of acidaemia and continued renal losses. To monitor potassium levels, they should be measured after initial fluid resuscitation and 6–8 hours after initiation of insulin therapy (Battaglia & Steele, 2016). If, after continuous potassium supplementation has been administered, the patient remains hypokalaemic, it is recommended that magnesium levels are checked. If there is a depletion in magnesium, potassium levels will not normalise (Battaglia & Steele, 2016).

Shifts in phosphorus will occur in the same way as potassium so, as the body is being treated for DKA, it will lose phosphorus and this will need to be replaced. Testing may not initially show hypophosphataemia as it is often most marked 1–2 days after initiation of insulin therapy, as phosphorus translocates into an intracellular location (Boag, 2012). The patient will need to have the phosphate levels checked every 6–12 hours during the first 48–72 hours of treatment (Boag, 2012).

**Table 4.** Potassium supplementation requirements according to serum potassium (VetsNow, 2013).

| Serum potassium (mmol/litre) | Potassium chloride to be added (mmol/litre fluids) |
|---|---|
| >5.5 | Do not add |
| 4.1–5.4 | 20 |
| 3.1–4 | 30 |
| 2.6–3 | 40 |
| 2–2.5 | 60 |

Hypophosphataemia will lead to intravascular haemolysis and supplementation will be required when plasma levels drop below 0.35 mmol/l (VetsNow, 2013).

Intravenous bicarbonate therapy is controversial, as acidosis will mostly improve with insulin and fluid therapy alone as ketones are metabolised (Battaglia & Steele, 2016). However, if the pH is <7.1 and not showing an upward trend or responding to insulin administration, bicarbonate supplementation can be administered via CRI, providing it is possible to measure blood pH (VetsNow, 2013). The dose of bicarbonate can be calculated as follows:

**$NaHCO^3$ – (mmol/l/hour) =**
**base deficit × 0.3 × weight (kg)**

Boag (2012) recommends giving one third to one half of this dose by slow intravenous infusion over 15–30 minutes. The acid–base status can then be reassessed on completion of the infusion. The remainder can then be added to the patient's fluids and given over a few hours, if necessary, while continuing to monitor acid–base and electrolyte status (Boag, 2012). When the pH reaches 7.2, the bicarbonate should immediately be stopped to prevent iatrogenic metabolic alkalosis (Battaglia & Steele, 2016).

## Nursing care

The role of the nurse in the care of a DKA patient is essential for an effective recovery. The patient is likely to be in a critical condition and will require almost continuous monitoring, whether that be recording vital signs or taking blood samples for glucose and electrolyte monitoring. Ideally, the patient will be placed in an intensive care facility with 24-hour nursing availability, as these patients are at a greater risk of deteriorating. Haskey (2015) discusses the use of a four-step nursing process to maximise the care of the critical patient: assessment, planning, implementation and evaluation. The steps are used on a continuous cycle as often as needed. In more critical patients, where drug therapy response is key, this process may be used every 15 minutes.

One of the key vital signs to be monitored is the respiratory rate, as it will give an indication of the acidosis compensatory mechanisms. When the body goes into acidosis it has three mechanisms for compensation: buffers, respiratory compensation and renal compensation. When the respiratory system starts compensating for acidosis you can expect to see increased ventilation leading to decreased partial pressure of carbon dioxide. This increase in respiratory rate should not include an increase in respiratory effort and therefore dyspnoea should be viewed as a separate clinical sign. It will be common for the patient to have an increased respiratory rate on presentation and this

should be monitored throughout the treatment process to ensure the patient shows signs of improvement rather than deterioration.

As with all patients, cardiac auscultation is essential. Heart rate and rhythm can change depending on varying factors. For example, a patient with increased blood potassium needs to be monitored, in particular, for signs of bradycardia. The auscultated heart rate and rhythm should always be compared with the pulse rate and rhythm to assess for pulse deficits indicating cardiac arrhythmias such as atrial fibrillation (Haskey, 2015). Any pulse deficits noticed should be confirmed with an ECG.

Adequate nutrition for the DKA patient is crucial to the recovery process but may prove challenging to maintain. Patients with DKA are often anorexic on presentation, with appetite not usually returning until ketoacidosis is controlled (Boag, 2012). Where possible, it would be recommended to maintain at least the resting energy requirements (RER) of the patient. This can be done through any means, from hand-feeding to tube-feeding (Haskey, 2015).

Fluid therapy should be monitored and maintained to ensure the patient is receiving the correct volume of fluids, as fluid overload in particular can be a complication of therapy. Hydration status can be monitored by evaluating the factors laid out in **Table 3**. Haskey (2015) states that with regards to fluids, 'ins' should match 'outs'. The veterinary nurse can perform 4- to 8-hourly urine measurements to ensure the patient is not being over- or under-hydrated. The urine can be measured from a urine collection bag if a urinary catheter is in place or by simply weighing the urine production on bedding, incontinence pads or in litter. Regular weighing of the patient is a useful tool for indication that the fluids are meeting the patient's requirements. The fasted pet can expect to lose 0.5–1.0% of body weight per day. Abnormal weight gain indicates over-hydration and will need to be monitored closely alongside urine output. The nursing of the critical DKA patient can be intensive and challenging, but following the four-step nursing process will ensure thorough monitoring, and intervention as required, ensuring the highest standard of care for the patient.

## Conclusion

Boag (2012) states that prognosis for the DKA patient is guarded, with a study showing that long-term (>2 months) survival was only 12%. With that in mind, and the first 24 hours being the most critical stage, it is crucial for the veterinary nurse to monitor the patient for signs of deterioration and clinical symptoms associated with ketoacidosis, to actively participate in the care and changing treatment plans. The veterinary surgeon relies on the nurse to be able to monitor closely and report changes as necessary. The care of the DKA patient is often extensive and requires intensive care from the team involved. Nursing care is invaluable to patient recovery and is a good opportunity for RVNs and SVNs to apply the knowledge gained through study and to see a clinical difference being made throughout the treatment process.

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