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The Secrets to Managing Problem Diabetics Mini Series

Session Three: Diabetic Emergencies

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Study notes Session 3 - Diabetic emergencies

Ketoacidosis, hyperglycaemic hyperosmolar syndrome (HHS) and hypoglycaemia

Introduction

Some diabetic cases go like a dream but many are a real headache require considerable input of time and energy with many being a marathon rather than a sprint. This three part webinar mini-series aims to look at the potential causes, how they can be recognised and solutions that might be available. In the speaker's experience more problems are related to insulin dose, especially over-swing and fewer to intercurrent disease than previously suggested.

The final session is focused on diabetic emergencies, how we recognise and manage them as some their crisis will be the first time they present with their diabetes. Not identifying that they are having a diabetic crisis can have a profound effect on their prognosis.

Outline

- Spotting diabetic crises
- Diagnosis of ketoacidosis
- Managing the ketoacidotic dog
- Managing the ketoacidotic cat
- Approach to hyperglycaemic hyperosmolar syndrome (HHS) in dogs
- Approach to HHS in cats
- Hypoglycaemia – improving client awareness and in-clinic response

General indicators of emergencies associated with insulin abnormalities

The crisis event may be acute or peracute BUT usually chronic, non-specific signs have been present particularly weight and appetite change, exercise intolerance, weakness (table 1).

Table 1 – Key historical signs associated with diabetic crises.

Historical signs	*Hyperglycaemia*	*Hypoglycaemia*
PU/PD	Lethargy	Collapse
Inappetence or polyphagia	Anorexia	Seizures
Weight change	Adipsia	Muscle weakness
Intermittent GIT signs	Vomiting and diarrhoea	Ataxia
Waxing and waning vague illness	Hyperventilation	
Lethargy/exercise intolerance		

Diabetic ketoacidosis

DKA should be distinguished from diabetic ketosis that can be present intermittently in many diabetic dogs especially those with poorly stable DM. Specific management of the ketosis is rarely required and should resolve with improved diabetic control.

DKA pathophysiology

DKA develops as a results of inadequate insulin to meet the metabolic needs either because treatment dose is insufficient for the current status [NB status can change and this can precipitate crisis e.g. development of a UTI] of the case or the patient has newly diagnosed DM and no insulin has been given. However not all dogs or cats with DM develop DKA

- Contributory factors to development of DKA
    - Excess diabetogenic hormones
        - Glucagon, growth hormone, glucocorticoids
    - Fasting leads to increased hormone secretion
    - Dehydration leads to a further reduction in the ability to excrete glucose and H^+ ions
    - Intercurrent disease
- Hyperglucagonemia
    - Primarily responsible for ketonemia
    - Insulin deficiency with glucagon excess results in peripheral lipolysis leading to increased plasma fatty acid concentrations

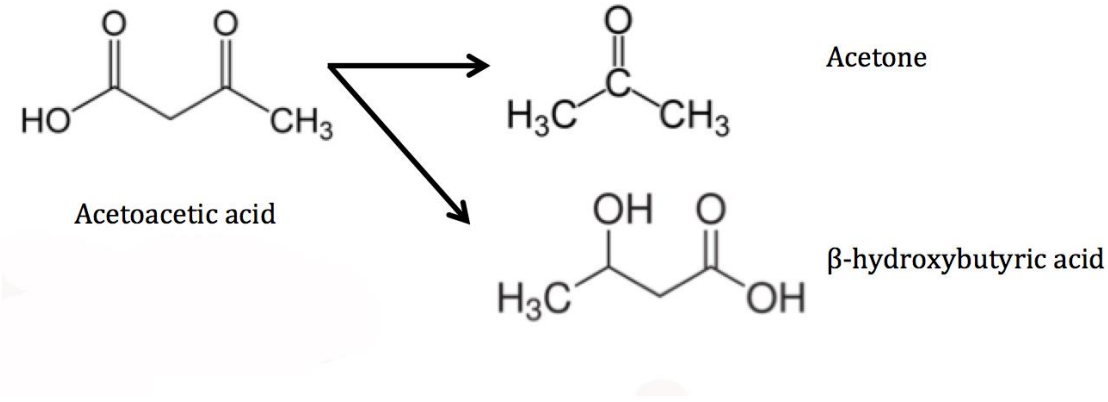
Under normal circumstances plasma fatty acids are metabolised to form acetyl coenzyme A in the liver and enter the triglyceride or citric acid cycle being metabolized to protein, glucose or glycogen. If glucagon levels are high plasma fatty acids form acetoacetyl coenzyme A that is metabolised to acetoacetic acid and thence to acetone and β-hydroxybutyric acid (ketones) (Fig. 1).

Ketone metabolism

Ketones are metabolized by many tissues as they serve as a short-term source of energy when glucose levels entering the cell are insufficient.

BUT in cases where production greatly exceeds utilization, metabolic acidosis and osmotic diuresis due to ketonuria occurs leading to dehydration, electrolyte depletion, vomiting and diarrhoea. The dehydration and hypovolaemia can lead to prerenal azotaemia and hyperosmolality. Derangements are progressive and will eventually result in death if not treated.

Figure 1 – Metabolism of acetoacetic acid



DKA presentation - dogs

Not all dogs with DKA will present as emergencies; cases will have a similar presentation to DM i.e. PU/PD, polyphagia, weight loss. In one study of 221 newly diagnosed diabetics, 15% had DKA. Of these 15%, 23 had concurrent disease.

- 20% UTI
- 20% HAC
- 15% acute pancreatitis
- 5% neoplasia
- 5% hypothyroid
- 5% other

Severe DKA signs are usually rapid in onset presenting with lethargy, anorexia, adipsia, vomiting and diarrhoea.

Physical examination

Generally non-specific signs associated with dehydration, acidosis and azotaemia – depression, dehydration, weakness, tachypnoea, abdominal pain, acetone breath (individual clinician ability to detect acetone variable). Other clinical signs due to secondary diseases (as above) may also be present.

DKA and HHS presentation – cats

Hyperglycaemia, hyperosmolar syndrome (HHS) is more commonly encountered in cats, distinguishing one form the other is important as it affects treatment decisions.

Risk factors

- Mean age of DKA cats is 9 (2-16 years) tended to be newly diagnosed diabetics compared to HHS with a mean age 12.6 ± 3.2 years with greater likelihood of already being treated for DM.
    - DKA cats tend to be younger than uncomplicated DM cases
    - Siamese cats may be over-represented
- Concurrent disease in 90% of DKA cats
    - Hepatic lipidosis, CKD, acute pancreatitis, infection and neoplasia
    - HHS cases – CKD, CHF and infection

Clinical signs

DKA - Most commonly PU/PD, lethargy, inappetence, anorexia, vomiting and weight loss
Less common - underweight, dehydrated, icterus and hepatomegaly

HHS – Commonly - ataxia, weakness, respiratory problems, neurologic signs (circling, pacing, unresponsive)
Less common - overweight, moderate to severe dehydration, respiratory compromise, hypothermia

DKA diagnosis

DKA will usually be suggested by a standard blood screen for emergency patients showing hyperglycaemia that is usually marked. Routine haematologic and biochemical changes have the features of most DM cases. Other common haematologic and biochemical abnormalities include

- Leucocytosis and haemoconcentration
- Pre-renal azotaemia
- Whole body potassium depletion but serum potassium can be normal to increased due to acidosis or AKI
    - Rarely severe life-threatening hypokalaemia
- Hyponatraemia due to diuretic-associated loss
- Hypophosphataemia – urinary loss vs. cellular shift

Definitive diagnosis requires presence of hyperglycaemia with acidosis and ketonemia or ketonuria.

Clinical pathology in cats

- DKA cases in addition are acidotic with ketonuria; Heinz bodies are common. Ketonemia will also be present although ketometers seem less able to distinguish between ketosis and DKA than in dogs.
- HHS cases show severe osmotic diuresis with marked hyperosmolality, azotaemia and variable sodium amounts. Anaemia is uncommon although this may reflect haemoconcentration.
- Hyperphosphataemia is more commonly seen in cats than hypophosphataemia as in people and dogs; however this does not mean that hypophosphataemia will not occur with therapy.
- Hypomagnesaemia in DKA seems common although of uncertain clinical significance.
- Sodium levels can be difficult to assess as hyperglycaemia can draw fluid into the extracellular space causing pseudohyponatraemia. Corrected sodium values are sometimes used to try and guesstimate true sodium levels.
    - 'Corrected sodium' – increase serum Na^+ by 1.5mmol/L per 5mmol/L increase in glucose

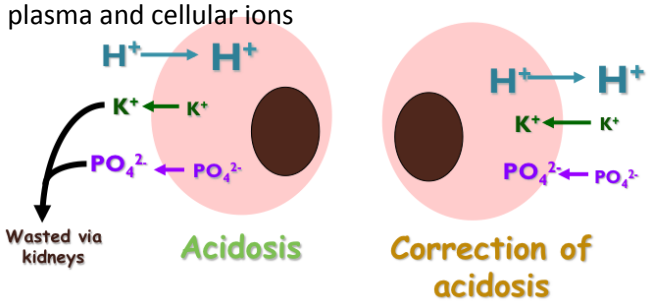
Acidosis

It is difficult to assess acid-base status without being able to measure plasma pH; to some extent it can be assumed in patients with high BG and ketonuria whose osmolality is normal.

Some DKA cases will show a marked acidosis seen as a low pH (<7.15), low bicarbonate and large negative base excess. pCO_2 will tend to be low in an attempt to compensate.

Acidosis can have profound effects on serum potassium and phosphorus levels (Fig. 2) giving a false impression of whole body changes.

Figure 2 – Effects of acidosis and its correction on plasma and cellular ions



Ketosis

Hand held ketometer (measure β-hydroxybutyrate) are widely available and inexpensive and give an opportunity to measure blood ketone levels to assess ketonemia rather than relying on ketonuria. There is a reasonable correlation between handheld meters and laboratory analysis of blood ketone levels however the precise reference ranges and cut-off points will vary with the meter used and as yet wide experience of their utility in general practice is lacking. In man β-HB levels often far exceed acetoacetic acid.

Suggested cut-off points (MediSense Optium, Abbott Laboratories, Oxon, UK) in dogs

- Low cut-off around 2.3mmol/L has high sensitivity
- High cut-off around 4.5mmol/L had high specificity

Ketone concentration <2·55 mmol/L (Precision Xtra, Abbott Laboratories, Oxon, UK) enable ketoacidaemia to be excluded in cats and should lead to redirection of differential diagnoses but some non-DKA cats can have quite high serum ketone levels.

The presence of ketosis does not of itself make a diagnosis of DKA (see hyperglycaemic, hyperosmolar syndrome). Ketones can be elevated in other diseases in dogs such as pancreatitis (Hurrell *et al* 2016) with values exceeding 2.3mmol/L in a one patient (1/42).

Traditionally ketosis is measured by looking for urinary ketones.

Urinalysis

- Glycosuria – usually ++++
- Ketonuria - dipstick react with acetone and acetoacetate so can underestimate the level of ketosis
    - Differential diagnosis of positive urinary ketone reaction
        - Out of date sticks
        - Fasting
        - Glycogen storage disease
        - Persistent fever
        - Persistent hypoglycaemia
        - Low carbohydrate diet
- USG poorly reflects true renal concentration due to effects of high levels of glucose on specific gravity; urine osmolality is a more accurate measure.
- Urine should also be submitted for sediment analysis and culture as around 20% of DKA cases are also likely to have a UTI.

Serum osmolality

DKA can have profound effects on serum osmolality due to dehydration and sodium wastage. Although not immediately available in-house estimation by calculation can help drive appropriate therapy (see page 10).

Osmolality ≅ 1.86 (Na^++ K^+) + glucose + urea +10

DKA and pancreatic disease

A study by Bolton *et al* (2016) in dogs suggested nearly ¾ of dogs have evidence concurrent pancreatic injury based on cPLi with fair agreement with abdominal ultrasound. There was a tendency for cPLi positive dogs to require longer hospitalisation (median 6 days vs. 4 days) with a poor survival rate (34/45 vs. 15/17) but these were not statistically significant. However pancreatitis should be considered in DKA dogs and may impact on costs and survival.

Treatment of severe DKA

Inappropriate therapy can hasten a patient's death so it is essential that each patient is assessed individually and has their own treatment plan formulated. Prior to therapy commencing (or as soon afterwards as possible) blood samples for diagnostics should be taken. DKA is a dynamic situation hence frequent monitoring of physical and biochemical parameters is crucial.

Aims of therapy

- Manage fluid balance
- Manage electrolytes and phosphorus
- Provide insulin and a carbohydrate substrate
- Correct acidosis
- Identify and treat precipitating causes and concurrent disease

Cats with DKA or HHS - American diabetes association guidelines are to treat with 0.9% saline for the first hour in patients with DKA and HHS and then use 0.45% saline if corrected sodium is high or within reference range. No studies have been conducted in cats to test the value of this approach.

Fluid therapy

Appropriate fluid therapy is the mainstay of DKA treatment. Preferably more than one large bore catheter should be placed. In patients that are severely dehydrated with circulatory collapse this can be challenging; start with a small catheter and as circulation is restored insert a 2nd large catheter at another site.

Then :-

- Estimate dehydration
- Estimate likely fluid losses –remember that the glycosuria means that osmotic diuresis will persist
    - Place a urinary catheter – ins and outs
- Choose fluids based on baseline electrolytes, acid-base and osmolality calculation. In general 0.9% NaCl or Hartmann's with added potassium, phosphorus and glucose as required is appropriate.
    - If the patient is hyperosmolar hypotonic saline may be required (<0.45% NaCl) - mix half a bag of 0.9% NaCl with half a bag of 5% dextrose.
- Assess response
    - Clinical status, urine output, bodyweight, central venous pressure if available and a central line has been fitted.

If high fluid rates are being used, repeat electrolytes, phosphorus, acid-base, urea & glucose in 2 hours as these can change dramatically through cellular shifting and renal excretion.

Fluid rate

IVFT should aim to meet hourly demands + replace 50% of the estimated deficit over 2-4 hours and the remainder of the deficit within 24 hours.

Example - 8kg diabetic miniature poodle with DKA Estimated 10% dehydrated = 800ml deficit Replacement requirement 100-200/hour Osmotic diuresis means producing 5ml/kg/hr urine Insensible losses @ 1ml/kg/hr Total fluid requirement = 148-248ml/hr (9=15 x maintenance!)

It is essential to monitor urine output especially in DKA cases with marked azotaemia as acute kidney injury may have occurred (be occurring) and is likely if urine output is low (<2ml/kg/hr). High fluid rates in these patients can lead to volume overloading.

Electrolyte balance

Sodium levels will be managed by fluids and kidneys alone. Due to electrolyte shifting associated with acidosis (Fig. 2) high serum potassium may still reflect a whole body deficit associated with renal wasting (unless patient has AKI) hence potassium supplementation of the fluids is required. Potassium supplementation of the fluids should be based on plasma levels and fluid rate.

It is the total amount of potassium that is being given that matters not the fluid concentration. In general potassium supplementation to the patient should not exceed 1mmol/kg/hour.

Example - 8kg diabetic miniature poodle with DKA estimated 10% dehydrated Amount of potassium given will depend on the speed with which fluid deficits is corrected Moderate amount of potassium added to fluids e.g. 40mmol/L 50% of deficit over 4 hours – (40 x 0.148)/8 = 0.74mmol potassium/kg/hr 50% of deficit over 2 hours – (40 x 0.248)/8 = 1.24mmol potassium/kg/hr

Managing phosphorus

Often forgotten in DKA patients, unmanaged hypophosphataemia can cause severe complications resulting in haemolytic anaemia if phosphate drops below approximately 0.5mmol/L. Supplementation should be started once phosphate falls below 0.6mmol/L

- Dose rate 0.01-0.03mmol/kg/hr
    - Options
        - Intravenous potassium phosphate (Fresenius Kabi 100mmol/500ml)
        - Foston - 2.5mmol/mL [approx.]

Insulin therapy

Reducing BG is important but rapid reduction can lead to profound electrolyte, acid-base and osmolality changes; hyperglycaemia *per se* is not life threatening so the aim is to SLOWLY decrease BG to <15mmol/L over the first 8 hours of treatment. Insulin should be given IM or IV as SC will probably be ineffective due to poor cutaneous circulation.

The author prefers IM administration as it is less complicated and less prone to error in the midst of a DKA crisis. A loading dose of 0.2iU/kg of neutral insulin should be given followed by 0.1iU/kg neutral insulin q1-2hr. A recent study (Gallagher *et al* 2015) compared low-dose CRI neutral insulin for managing DKA cats with SC glargine – IM neutral insulin combination in 16 randomised cases. Time to resolution of hyperglycaemia, hospitalisation and normalisation of acidosis and ketosis was significantly shorter in the SC/IM group.

If IV CRI is to be used it should be given via a separate syringe driver as this allows changes in IVFT rate without changes rate insulin is administered or necessitating a new bag of fluid to be made up.

- Dilute insulin in 0.9% saline to a standard concentration e.g. 0.5iU/ml and alter rate (table 2) of administration to alter dose of insulin given.
- Hypoglycaemia is potentially a greater risk than using IM therapy.

Better outcomes have been achieved when higher doses of insulin have been used.

Table 2 – Suggested administration rates (ml) of 0.5iU/ml solution of insulin according to weight & BG level

BG (mmol/L)	5kg dog/cat	10kg dog	20kg dog	30kg dog
>15	1-2	2-4	4-8	6-12
12-15	1	2	4	6
9-12	0.75	1.5	3	4.5
6-9	0.5	1	2	3
<6	Stop	Stop	Stop	Stop

Managing insulin therapy

$$H_2O + CO_2 \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-$$

- Monitor blood glucose hourly
  - Aim to decrease BG by <5mmol/L/hr
- Once insulin below 15mmol/L move to q4-6hr administration
- Move to longer acting insulin when patient alert and ketosis resolved (can take 48 hours to occur).

Glucose therapy

As glucose levels fall the risk of hypoglycaemia increases and glucose should be added to the infusion solution (table 3). As insulin effects will last longer than glucose will persist in the circulation, glucose therapy should continue for at least 2 hours after insulin therapy has been stopped.

Table 3 – Suggested rates of glucose inclusion to IVFT with change in BG

BG (mmol/L)	% glucose in fluids	ml of 50% glucose to 1L
>15	0	0
12-15	2.5	5
9-12	2.5	5
6-9	5	10
<6	5	10

Managing acid-base disturbances

Acidosis is can be profound with a pH <7.0 is a being a poor prognostic indicator for survival. In general, the kidneys are good at sorting out acidosis so where possible IVFT alone should be used. However if the acidosis is profound (pH < 7.20 and/or bicarbonate <11mmol/L), bicarbonate may be required. Bicarbonate is available as 8.4% $NaHcO_3^-$ solution that is equivalent to 1mmol/ml of bicarbonate.

There are two approaches to therapy; the author prefers to give a standard amount and monitor the response as despite the formulae that are available, the true effect of adding bicarbonate to the system can be difficult to predict.

- Give 0.5-1mmol/kg of sodium bicarbonate over 20 minutes slow IV regardless of level of acidosis
  - This level is also safe is acidosis cannot be measure but is assumed to be present and severe enough to warrant therapy
- Calculated dose (in mmol) = Bodyweight (kg) x 0.4 x (12-patient bicarbonate)

As with BG, it is important to correct acidosis slowly due to the potential adverse effects of rapid correction

- Metabolic alkalosis
- Paradoxical cerebral acidosis
  - Correction of acidosis generates CO_2
  - Most of CO_2 removed by lungs

    - Some diffuses across BBB and recombines with water to produce H^+ ions

    - Progressive worsening neurologic signs despite therapy
- Rapid falls in potassium and phosphate to life-threatening levels

Identifying and treating precipitating factors
Most DKA patients will have other issues; identifying these issues will help long term management but may be of limited value short term. Once stabilization is under way, consider -

- Abdominal ultrasound, thoracic radiographs
- IV antimicrobials to treat UTI
- Evaluating patient for HAC of limited value in acute phase

Prognosis
Outcome of DKA in dogs
With good management the prognosis for DKA dogs is good, however, severe cases that present late in the course of their disease can die despite intensive support.
Results from a large study of DKA dogs

- 70% of dogs survived to be discharged
- Median hospitalization was 6 days
- Negatively survival factors
    - Lower ionized calcium
    - Lower haematocrit
    - Lower venous blood gas
    - Larger base deficit

Outcome of DKA and HHS in cats

- 60-70% of DKA cats survived to discharge with median duration of around hospitalisation of 5 days; 40% had recurrent episodes
    - Recent published survivals have been
        - 50% of 10 cats (Bollinger & Moore 2015)
        - 61% of 93 cats (Cooper *et al* 2015)
        - 59% of 16 cats (Gallagher *et al* 2015)
- HHS seems to carry a worse prognosis with only 35% surviving to discharge

A recent study of 93 cats with DKA (Cooper *et al* 2015) found that a poor outcome was predicted by higher BUN/creatinine, serum magnesium and bilirubin. Treatment with higher concentrations of insulin tended to improve outcome.

Hyperglycaemic hyperosmolar syndrome [HHS] in dogs

HSS is an unusual but severe complication to DM in dogs reported in up to 5% of DM cases (Trotman *et al* 2015); cases show severe hyperglycaemia and azotaemia resulting in marked hyperosmolality causing CNS depression, lethargy, hypovolaemia. Cases present with severe dehydration, depression, weakness and shock. Calculated osmolality will depend on plasma sodium that can be anywhere from low to high. See below for corrected sodium calculation. Some authors have divided these cases into ketotic and non-ketotic cases but in reality the ketotic cases fit more closely with severe DKA cases.

Pathophysiology

The development of HHS is attributed to three factors

1. Decreased insulin utilisation and glucose transport
2. Increased gluconeogenesis and glycogenolysis
3. Impaired renal excretion of glucose

The condition is characterised by a lack of ketosis possibly due to inactivation of β-oxidation of incoming free fatty acids and/or enhanced gluconeogenesis in the liver due to excess glucagon. These factors associated with severe dehydration (usually in excess of 8%) and reduced urine production results in marked hyperglycaemia. Neurologic abnormalities occur due to the effect of serum sodium concentration and hyperosmolality on the brain.

Levels of sodium that are normal to elevated reflect substantial cellular dehydration. Due to the effects of hypovolaemia and hyperglycaemia on sodium levels, formulae to calculate corrected sodium have been advocated but the clinical utility of this calculation is unclear although it may serve to highlight the degree of free water deficit.

- 'Corrected sodium' – increase serum sodium by 1.5mmol/L for every 5mmol/L increase in glucose

i.e. if a dog's sodium is 150mmol/L [ref: 139-154] and glucose 45mmol/L [ref: 3.5-5.5], corrected sodium is 162mmol/L

Osmolality can be directly measured by a variety of methods by most external laboratories, an approximate calculation can be made using the following formula

Osmolality ≅ 1.86 (Na^+ + K^+) + glucose + urea +10

The reference range for cats and dogs is 290-310mOsm/kg

Examples

DKA dog : K^+ – 4.8mmol/L; Na^+ – 130mmol/L; glucose 32.5mmol/L; urea 18.2mmol/L

Calculation

1.86(4.8 + 130) + 32.5 + 18.2 + 10 = 311mOsm/kg [within reference interval]

HHS dog: K^+ – 6.5mmol/L; Na^+ – 145mmol/L; glucose 55mmol/L; urea 40mmol/L

Calculation

1.86(6.5 + 145) + 55 + 40 + 10 = 356.8mOsm/kg [markedly above reference interval]

Treatment

- Careful choice of fluids and rate to prevent cerebral oedema
  - Even if sodium is high, sodium containing fluids are required; initially ≥ 0.45% sodium
- Slow reduction of glucose at 3-5mmol/hr
- Re-establish urine output to > 2ml/kg/hr
- Regular (initially every 1-2 hours) monitoring of electrolytes, glucose, urea, (phosphate)
- Hourly assessment of level of consciousness & mentation in case cerebral oedema occurs.

Prognosis

Poor prognostic indicators (Trotman *et al* 2015) were abnormal mental status and low venous pH. 62% of cases in this study survived to discharge.

Hypoglycaemia

Hypoglycaemia can result from a variety of disease states, most commonly associated with an excess of insulin either given exogenously or endogenous (insulinoma – relatively common in dogs; very rare in cats). Less commonly hypoglycaemia will be associated with decreased production or drug-induced. The most common cause on most biochemical screens is a delayed separation of serum or plasma resulting in white cell metabolism of glucose.

- Decreased glucose production
    - Severe liver disease – toy breeds
    - Hormonal – hypoadrenocorticism, GH deficiency, hypopituitarism
    - Fasting, malnutrition, pregnancy, extreme exercise
    - Sepsis
- Drug-induced – rare e.g. β-blockers, amitriptyline

Insulin overdose

Robust procedures should be in place in practice to prevent overdose of insulin being given to hospitalised patients. Even so mistakes will and do occur most commonly due to misreading the prescribed dose. Similarly mistakes can also be made by clients particularly following a dose change. To some extent this can be lessened by the use of insulin pens. Cats and small dogs tend to be at higher risk due to the low total volume of insulin being given. Cats may be more prone to relative insulin overdose as transient diabetes, variations in the level of insulin resistance and glucose toxicity are more prominent features of their disease making variation in the dose of insulin required more likely over time.

Common causes of insulin overdose

- Using 40iU/ml syringes with 100iU/ml insulin
- New insulin bottle – if the previous insulin supply has been damaged and is much less effective leading to significant increase in dose, when a new bottle is started this can lead to a relative overdose.
- Insulin given and patient refusing to eat
- Insulin dose high due to resistance that now has resolved – e.g. increased dose given associated with a UTI that is then treated with antibacterials, post-seasonal diabetes.
- Transient diabetes in cats

Insulin overdose should always be suspected in a collapsed diabetic patient; if there is any doubt glucose should be given (e.g. patient still at home) until BG can be measured.

Risk factors for insulin overdose in cats

- Neutered male cats >12 years old (normal diabetic population!)
- Heavier cats >5.8kg
- Higher insulin doses >6iU/dose

Insulin-induced hypoglycaemia (Somogyi overswing)

Insulin-induced hypoglycaemia is probably a relatively common phenomenon and accounts for a significant number of unstable diabetics. Excess insulin dose causes BG to drop below the reference range. This process can happen very rapidly (10-20 minutes) making it difficult to document in some patients using standard glucose curves.

Following hypoglycaemia, gluconeogenic hormonal environment leads to apparent insulin resistance for up to 2-3 days. BG measurements made during this period when counter-regulatory hormones are high tend to suggest insulin resistance so dose is increased creating and ever lowering nadir until a hypoglycaemic crisis occurs.

Common signs of insulin overdose (hypoglycaemia)

- Lethargy, dullness and anorexia
- Seizures
- Recumbency
- Shaking
- Vomiting
- Ataxia

Managing a hypoglycaemic crisis associated with insulin overdose

Hypoglycaemia will cause rapid irreversible brain damage. Aggressive early treatment is therefore essential; oral glucose can be absorbed through the buccal and oesophageal mucosa. All owners of diabetic dogs on insulin should be aware of the clinical signs of hypoglycaemia – tremors, disorientation, visual disturbance, behavioural changes, weakness , ataxia, collapse and seizures

Patient at home

- Give glucose, honey, Ribena concentrate, Lucozade, jam, sugar water and bring to surgery immediately – equivalent of about 1g/kg
    - Make sure that these are not low sugar preparations
- Ideally all owners with diabetic dogs should have glucose tablets (about 50g/tablet) or Glucogel (Hypostop) (10g/tube or 30g/bottle) at home and be aware of what dose to give (0.5-1g/kg).

At surgery

- Immediate IV access and 0.5-1g/kg glucose (1-2ml 50% glucose/kg) over 5-10 minutes.
    - If immediate IV access not possible give Glucogel
- Continue on 5% glucose saline for at least the next 24 hours. Initially monitor the blood glucose every 30-60 minutes.
- If there has been a massive overdose of insulin dose (especially in the practice) or non-responsive hypoglycaemia consider glucagon.

GLUCAGON (GlucaGen Hypokit)

- Effect in 30 min
- Persists for about 90 min
- Initially give a 50 ng/kg bolus IV then
- CRI at 10–15 up to 40ng/kg/minute
    - 1mg vials with prefilled syringe containing water for injection
    - Inject reconstituted 1mg into 1 litre of 5% dextrose (0.9% saline if 5% dextrose not available) to make a 1µg/ml solution.
        - Give 0.05ml/kg of solution as IV bolus

Conclusions

DKA is the most common endocrine emergency that is likely to be seen in first opinion practices. As most are undiagnosed diabetics DKA should be considered as a potential cause for collapsed, dehydrated cats or dogs especially with GIT signs appropriate treatment carries a good prognosis for recovery. Although an uncommon cause of weakness, collapse or seizuring, failure to recognise hypoglycaemic seizuring will result in a poor response to anti-seizure medication and, if uncorrected, death of the patient.

- Always check if emergency cases are known diabetics
- Glucose estimation is mandatory in all emergency cases

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