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Managing Diabetic Patients Mini Series

Session Two: Nursing the Diabetic In-Patient

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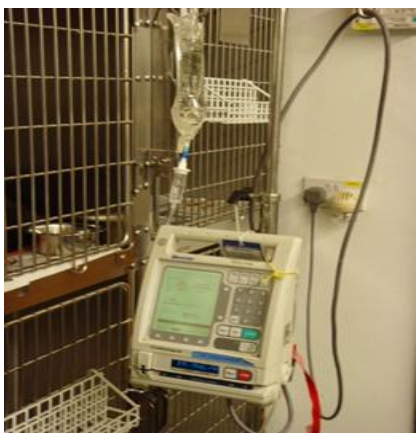


Hypoglycaemia.

Clinical signs of hypoglycaemia should be explained to pet owners at the time of the initial diagnosis of diabetes mellitus; though the signs will differ from patient to patient.

Signs can include:

- Lethargy
- Ataxia
- Muscle twitching
- Severe seizures
- Disorientation
- Lack of responsiveness
- Coma

Peripheral blood sampling can be very difficult in a hypoglycaemic animal, with signs of shock being displayed and peripheral vasoconstriction. Treatment of the clinical signs is urgently required, followed by close monitoring and investigation of the cause of the hypoglycaemia. Administration of glucose followed by a meal of highly digestible food is required.

Care is required when administering intravenous glucose as it is an irritant when administered in high concentrations. All glucose solutions should be diluted with sterile saline or water for injection, to make a 10-20% glucose solution for administration via a peripheral vein. Higher levels can be given if a central line is utilised. Glucose supplementation is more effective when a loading dose is administered followed by a continuous rate infusion (CRIs). As with all CRIs, they aid to remove the peaks and troughs of repeated boluses.

It is important to actively encourage the animal to eat after glucose administration. The initial cause of the lower blood glucose level can still be ongoing, and therefore a more sustained supply of glucose can be required. A highly digestible diet should be fed as this will provide simple carbohydrates to the animal a lot quicker, than a high fat diet. Dextrose powder can be mixed into the diet to increase the

simple sugar contribution. High fat diets need to be avoided as this will cases delayed gastric empting. The ingested food needs to reach the small intestine in order for the simple sugars to be absorbed. A low fat, low fibre, highly/easily digestible diet should be used in order to get the ingested simple sugars into the small intestine as quickly as possible.

After a diabetic has experience a period over low blood glucose levels, the body produces other hormones that will cause an increase in blood glucose level. These Somorgyi over swings are primarily due to too much insulin being administered, (maybe as the overall requirement is decreased through many different reasons). Performing a glucose curve after a hypoglycaemic episode will show an increased blood glucose due to these compensatory mechanisms.

Diabetic Ketoacidoisis.

Can be seen in those animals that are previously thought to be healthy, or those that have been previously diagnosed with diabetes mellitus. Cats can undergo waxing waning levels of insulin (and thus requirements). If a diabetic dog (that has been previously stable) presents with DKA, we need to do further investigations to what mechanisms of insulin resistance are present.

Clinical Signs can include:

- Polyuria/polydipsia
- Anorexia
- Vomiting and/or Diarrhoea
- Depression
- Weakness or Collapse
- Poor body condition
- Hepatomegaly
- Acetone smell on breath
- Deeper more rapid respiration reflecting metabolic acidosis.

Diabetic Ketoacidosis (DKA) can arise in an animal with previously diagnosed diabetes mellitus (DM) or it can appear to occur suddenly in an animal that the owner thought to be healthy. For DKA to develop there is usually a triggering condition, as shown in the table below. This triggering condition then causes the glucagon:insulin ratio to increase. Glycagon promotes glycogenesis and the formation of ketoacids. Insulin is also required for metabolism of ketones to carbon dioxide and water. When there is a low level of ketone production in uncomplicated DM, the ketones can be metabolised and do not build up to the point of which they cause clinical signs.

Bacterial Infections
Any significant infected focus but especially:
Urinary tract infection – upper and lower
Prostatitis
Pneumonia
Pyoderma
Otitis externa
Severe gingivitis/oral abscesses from tooth root infections
Inflammatory Disease
Pancreatitis
Endocrinopathies or physiological endocrine changes
Hyperadrenocorticism (cats and dogs)
Acromegaly (cats and dogs, though different mechanisms)
Hyperthyroidism (cats)
Hypothyroidism (dogs)

Phaeochromocytoma
Dioestrus phase of oestrus cycle
Iatrogenic causes
Steroid therapy – including intra-aural or ocular

Table: Conditions that can trigger DKA through insulin resistance.

Initial Assessment of DKA Patients.

The initial assessment of these patients needs to completed as rapidly as possible. Parameters to be measured include:

- Packed cell volume (PCV) alongside total solids/proteins
- Electrolytes – sodium, chloride, potassium (and phosphorus)
- Renal function assessment – urea, creatinine and phosphorus
- Blood gas analysis
- Blood ketone levels
- Urine analysis
    - Specific Gravity
    - Dipstick analysis – assess for ketones
    - Sediment examination
    - Submit for culture

Blood serum ketones should be tested for, but many practices don't have the facilities in order to do this. Urine dipsticks can be used to measure ketone levels, but do not measure the most predominant ketone body (β- hydroxybutyrate), therefore false negatives can be seen. The addition of hydrogen peroxide to the sample to oxidise the β-hydroxybutyrate to acetoacetate will enable the urine dipstick to measure all of the ketone bodies.

Small handheld ketone meters are becoming more readily available, and are fairly inexpensive. Reference ranges for ketones are often quoted as:

- Fed State: 0.1mmol/l
- Overnight fast: 0.3-0.7mmol/l
- Metabolic foods: 1-3mmol/l
- Diabetic Ketoacidosis: >15mmol/l

The overnight fast has a higher ketone level than those fed a normal diet, as the body is using stored body fats as an energy source. Metabolic diets includes Purina DM and Hills m/d diets.

Management of DKA Patients.

The initial management of DKA patients is to provide insulin in order to reduce the hyperglycaemia and promote ketone metabolism whilst correcting the intravascular volume, correcting hydration levels and any electrolyte imbalances.

Fluid Therapy.

Fluid therapy is a vital aspect of the initial emergency treatment of DKA. The aims of fluid therapy are to:

- Address dehydration issues.
- Correction of Acid base balance.
- Create a means in which to deliver medications, glucose, electrolytes.

Current recommendations are to address dehydration and correct potassium prior to administration of insulin.

In order to administer fluids we need to ensure that we have correctly calculated the correct requirement for the patient. The percentage of dehydration and the calculated deficit needs to added to the maintenance requirements of the patient.

Percentage of dehydration	Clinical Signs
<5%	No obvious outward signs Concentrated urine
5-8%	Slightly prolonged CRT Slight tenting of the skin Mucous membranes feel tacky Third eyelid visible
8-10%	Sunken eyes Prolonged CRT Obvious tenting of the skin
10-12%	Oliguria Tented skin remains in place Clinical shock can be experienced
>12%	Progressive shock Coma and death.

The level of hydration of the animal needs to be accessed. This can be completed through clinical examination, looking at haematology parameters such as PCV and Total Protein levels. Once the fluid requirements have been calculated, then need to determine if a shock rate bolus is required. Need to

also ascertain whether additional IV access lines are needed. Especially if potassium and insulin are going to be administered intravenously.

When using PCV to access whether the animal is dehydrated it needs to be done in conjunction with Total Protein levels. TP can be measured on a urine refractometer. Depending on levels will help guide with assessment with fluid requirements.

- Increase in PCV and TP = dehydration
- Decrease in PCV and TP = aggressive IVFT, haemorrhage.
- Decreased PCV and normal TP = possible increased destruction of RBCs.
- Increased PCV and decreased TP = dehydration with protein loss, e.g. gastro enteritis.

Shock rate for isotonic crystalloids will vary greatly. The values below are guidelines and are per hour. A bolus for a defined period of time is then administered, for example 15mins. The animal is then examined again and a discussion made to whether another bolus given whether the animal is moved onto more maintenance requirements/rates.

- Shock dose = 60-90ml/kg (dog) or 40-60ml/kg (cat)
- Moderate hypoperfusion = 30-50ml/kg (dog) or 10-20ml/kg (cat).
- Mild = 10-20ml/kg (dog) or 5-7ml/kg (cat).

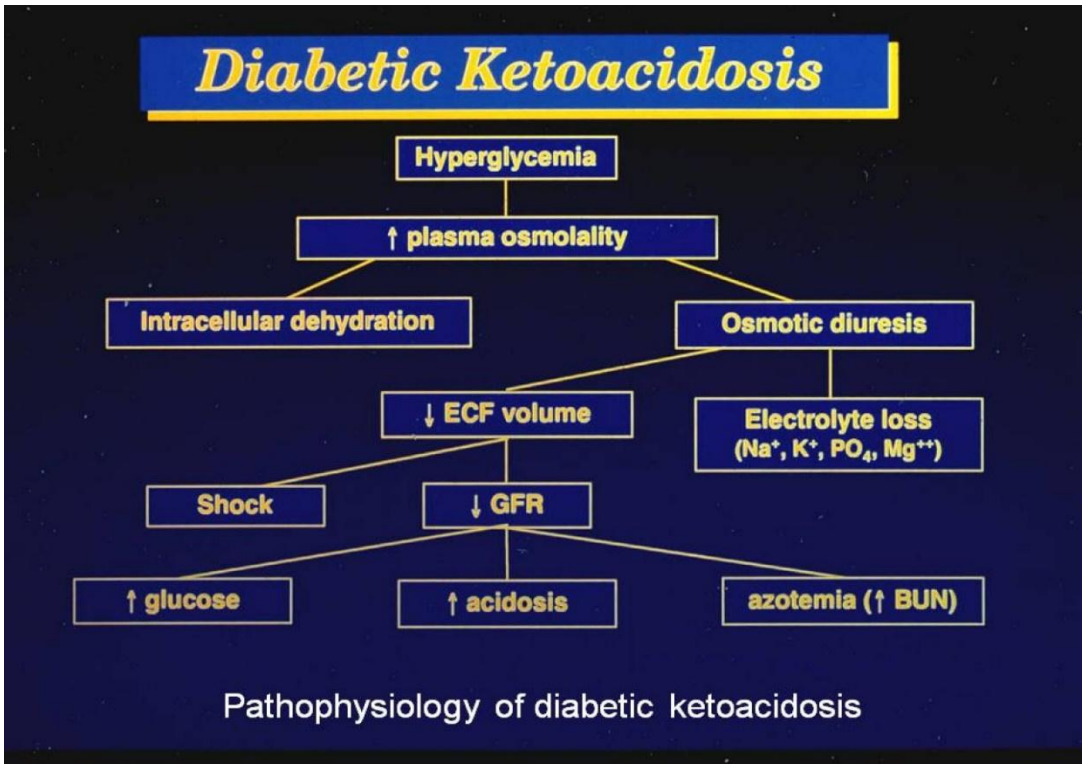
A fluid pump, syringe driver or burette should be utilised in order to achieve correct volumes being administered.

When calculating total fluid requirements we need to include:

- Maintenance fluid needs (in a non-diabetic) approx. 50mls/kg/day
- Replacement of ongoing losses associated with polyuria, typically 25ml/kg/day
- Correction of volume deficits over a period of 24hours (typically 50-100ml/kg/day)

The rate of fluid administration depends on the clinical assessment of hydration status, degree of shock, and the presence of concurrent disease, which could limit the rate of infusion.

In many cases Hartmanns solution (lactated Ringers, LRS, No 11) should be used, in order to aid with acidosis. In severe cases sodium bicarbonate can be administered, but this should only be done with the use of blood gas analysis analysers and syringe pumps/ syringe drivers. Dial in giving sets are not accurate enough, and are dependent on positioning of the animal, if administering sodium bicarbonate. In many cases the acidosis will be corrected as the ketone levels are decreased. This occurs as soon as fluid administration occurs as the total circulating volume is increased and therefore has a dilution effect on the amount of ketones and hyperglycaemia.



Electrolytes.

Many DKA patients can initially present with a ‘normal’ potassium level at initial assessment. Diabetes and especially DKA cause total potassium and phosphate depletion because of a shift of these electrolytes out of the cells into the serum to replenish losses and to help off-set acid-base imbalances. IVFT (given during the treatment of DKA) will further dilute / decrease electrolyte concentrations as will insulin-mediated uptake of phosphate and potassium by the cells, and renal

losses. Potassium and phosphate levels needs to be monitored prior to administration of treatment and throughout.

	Typical Guidelines	Guidelines for DKA
Serum Potassium (mEq/L or mmol/l)	KCl (mmol/l) to add to 1L of fluids	KCl (mmol/l) to add to 1L of fluids
>5.0	Wait	Wait
4.0-5.5	10	20-30
3.5-4.0	20	30-40
3.0-3.5	30	40-50
2.5-3.0	40	50-60
2.0-2.5	60	60-80
<2.0	80	80

Total hourly potassium, administration should not exceed 0.5mEq/kg bodyweight. Figures from Syme (2016).

Phosphate levels should be monitored alongside the other electrolytes (Na, Cl and K). Phosphate ions follow the same path as the potassium ions. The high extracellular glucose levels encourage movement of water, potassium and phosphate out of the cells. When fluid replacement and insulin therapy, the electrolytes are taken up into the cells. Hypophosphataemia often becomes most severe on the second day of therapy.

Phosphate supplementation is recommended if the phosphate concentration drops below 0.35mmol/l. The phosphate supplementation rate suggested in many formularies (0.01-0.03mmol/kg/h over 4-6

hours) is often insufficient in these patients and doses up to 0.12mmol/kg/h for 12-48hours may be required. Regular monitoring of phosphate (every 4-12hours) is necessary, with dose dependent adjustments. Take care as many phosphate supplements are supplied as potassium phosphate and you will need to take care of not over supplementing the potassium.

Severe hypophosphataemia can lead to haemolysis. In cats haemolytic anaemia will occur when phosphate concentrations decrease to less than 0.3 to 0.45mmol/l. Always check for haemolysis in the sample, especially on the second day where phosphate levels can drop severely. In some severe cases blood transfusions can be indicated. Figures of PCVs dropping below 20% being a cut-off point.

Insulin Therapy.

The main therapy for DKA patients is through insulin, and this can be achieved in two different formats dependant on the equipment that is available in practice.

Intramuscular Insulin:

- Begin treatment with 0.2IU/kg intramuscular bolus of neutral (soluble/regular) insulin.
- Repeat intramuscular injections of 0.1IU/kg hourly according to blood glucose measurements, measurements need to be kept within 8-15mmol/l.
- If the blood glucose drops below 8mmol/l, add 5% dextrose to the intravenous fluids, monitor blood glucose and continue insulin therapy if possible.
- Use neutral/soluble/regular insulin for the initial therapy or until the animal begins to eat reliably. Once eating the animal can be changed onto maintenance stabilisation using a longer-acting insulin.

Intravenous Constant Rate Infusion (CRI):

- Mix the neutral/soluble/regular insulin with fluids such that it will be delivered at a dose rate of 2.2IU/kg/day.
- Use of a syringe driver or fluid pump needs to be used in order to have accurate administration of the insulin. A flow rate of 1-2mls/kg/hr can be administered for the insulin, and addition fluid therapy can be run alongside.

- The insulin mixture needs to be protected from light, covered with aluminium foil, or bandage, and freshly made up every 24hours.
- As insulin binds to plastic tubing in drip lines, prior to administration the fluid mixture needs to be run through the line until a stable solution has been achieved (30-50mls expelled).
- Blood glucose needs to be checked after 1hour then every 1-2 hours thereafter.
- Dextrose can be used as required to maintain the blood glucose between 8 and 15mmol/l.
- Long acting insulin can be introduced when the animal starts to eat.

There is a danger that 'Piggy Backing' can occur with Neutral (Soluble) insulin. This is when the on-board insulin simply refers to the amount of insulin still circulating in the bloodstream that may still be working. Stacking or piggybacking insulin refers to when a patient gives more insulin before the previous bolus of insulin has finished working. This creates even more on-board insulin. When insulin stacking is done without care or proper understanding and direction from your doctor it can lead to serious episodes of hypoglycaemia (low blood sugar). Soluble insulin can last anywhere from one to four hours, so we do need to instigate careful monitoring of patients started on this type of insulin.

Feeding on In-Patients.

Once the patient is eating sufficient regular volumes of food it can then be changed on to a regular more long-acting insulin. Daily energy requirements for these patients needs to be calculated, alongside the daily volume / weight of diet to be consumed.

1. Calculate the RER of the animal

RER = 70 x (bwt kg)$^{0.75}$ for animals <2kg or >45kg, or 30 x (bwt kg) + 70

2. Add in the illnesses factor.

RER x Illness factor = kcal/day

3. Choose the specific diet, which is most beneficial for the patient, and the method of feeding.

4. Divide the energy content of the diet (kcal/ml or gram) by the energy requirement of the animal (kcal/day) to achieve the daily amount of food required.

5. Divide the total amount to be given in a day by the total amount of feeding wished to be given, or by maximum volume of each feed.

Illness factors are generally no longer used in practice, but do help to give you an ideal of what effect disease processes or recovery have on calorific requirements. Use your nutritional assessments to check whether sufficient calories are being consumed.

Anaesthetic Protocols for Diabetic Patients.

Management of blood glucose concentrations is the most important consideration when anaesthetising patients with diabetes mellitus. Owners of stable diabetic patients that are to undergo a procedure under anaesthesia should be recommended to give a half dose of insulin on the morning of the procedure. The procedures should be scheduled so that the diabetic patient is the first procedure of the day, so that it can be fed post procedure as soon as possible.

Following pre-medication a blood glucose sample should be taken for a baseline level. If the patient is already hypoglycaemic (blood glucose <4mmol/l) then intravenous glucose should be administered. Throughout the procedure if the blood glucose drops below 4mmol/l, administration of glucose should be instigated. A 5% dextrose drip can be used.

Intravenous glucose is highly irritant, and should be given diluted with saline or water. Supplementation is more effective when a loading dose is given followed by a continuous rate infusion. This will help reduce any peaks or troughs in blood glucose levels. On recovery from anaesthesia, monitoring of blood glucose should occur until the animal has recovered fully to in order to eat. A highly digestible (low fat) diet should be offered.

Pre-medication and use of alpha-2 agonists should be avoided as they cause an elevation in blood glucose during the procedure, and once reversed a refractory decrease in blood glucose level. Very careful monitoring needs to be utilised if alpha-2 agonists are therefore used.