Diabetes management: focus on role of veterinary nurses

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Michelle Donovan RVN, DipAVN(Medicine), discusses the part VNs have to play in stabilising and managing a patient’s condition.

DIABETES mellitus (DM) is an endocrine disease where a malfunction of the beta cells of the pancreas, which produce and secrete insulin, cause an imbalance of blood glucose levels.

The pancreas is found in the cranial abdomen, close to the stomach and duodenum. It is comprised of exocrine and endocrine cells. The endocrine pancreas consists of the Islets of Langerhans, which contain the beta cells that secrete insulin.

Diabetes mellitus is often seen in middle-aged and older entire bitches, dogs receiving progesterones for oestrus control and obese male cats. Burmese cats appear to have a predisposition to DM. Clinical signs are typically polydipsia/polyuria and polyphagia, and weight loss. This results from hyperglycaemia (the normal blood glucose range is 5-7mmol/l), glycosuria and osmotic diuresis.

Insulin induces cellular uptake of glucose from the blood, and its utilisation by tissues. When insulin is not produced, hyperglycaemia results. Excess blood glucose levels become too high for the renal tubular cells to resorb glucose from the glomerular ultrafiltrate, resulting in excretion of glucose in urine, resulting in glycosuria. Polyuria is caused by glycosuria, which induces an osmotic diuresis, resulting in dilute urinespecific gravity (USG). USG can be falsely increased by glucose in urine. Polyuria is compensated for by polydipsia, to prevent dehydration.
Weight loss in diabetic patients is due to the inability of the tissues to use glucose, causing the body to break down muscle, fats and protein. This causes weight loss due to catabolic metabolism. Ketones are produced during metabolism while this catabolic state is ongoing, and the ketones may build up and lead to ketoacidosis. The patient is polyphagic, regardless of hyperglycaemia, because cells are unable to use glucose. Therefore, the patient still feels hungry and the body feels it needs more food to compensate (Nelson, 2000).

**Type-I diabetes**

There are two types of DM. Insulin-dependent diabetes mellitus, also known as type-I DM, is most commonly seen in dogs. The beta cells of the pancreas fail to produce insulin, resulting in hyperglycaemia. Type-I DM can be caused by damage to the pancreas that is often immune mediated, and trauma, chronic pancreatitis and exhaustion of the beta cells from type-II DM.

**Type-II diabetes**

Non-insulin-dependent diabetes mellitus (NIDDM), also known as type-II DM, is most commonly seen in cats and entire bitches. This occurs when the beta cells do not secrete sufficient insulin to reduce the hyperglycaemia. The tissues are insulin resistant – therefore, the tissues are not using glucose. Over time, the demand on the pancreas to produce more insulin will exhaust the beta cells, which can lead to type-one DM. Type-two DM can be caused by obesity, transient DM in cats and high concentrations of hormones – for example, in the entire bitch, antagonism by progesterone results in beta cell exhaustion, whether endogenous or exogenous. Other examples of high concentrations of hormones are glucocorticoids (cortisol) and catecholamines (adrenaline), which antagonise the effects of insulin. Chronic pancreatitis in cats is now a more commonly recognised cause of DM at terminal stages, and is often associated with exocrine pancreatic insufficiency.

**Diagnosis**

The vet’s diagnosis is based on signalment, clinical signs, history and blood urine tests. In dogs, glycosuria and hyperglycaemia are usually present. If these signs are not present, an intravenous (IV) glucose tolerance test can be performed. The patient will be termed glucose intolerant if the normal glucose peak is high and it takes a long time for the glucose concentration to return to normal. The normal glucose range is 5-7mmol/l.

Cats become stressed easily, such as when taking a blood sample, which can cause stress hyperglycaemia and, possibly, glycosuria. A urine sample can be obtained at home, when the cat is more relaxed, and tested with a dipstick (Figure 1).

A serum fructosamine concentration will provide information about the average blood glucose concentration over approximately three weeks.
Patient management

The key to successfully managing diabetes mellitus is to first treat any underlying disease or complication. Spay entire bitches, treat complications such as Cushing’s and acromegaly, and treat any infection (especially urinary tract infections, which are common in DM due to increased amounts of glucose in the urine). Normal bodyweight must be established or maintained. Nurses can advise owners about nutrition; dietary requirements should be based on the caloric requirement for the ideal weight of the patient. Obese patients should gradually reduce weight at one to two per cent bodyweight per week. If the patient is underweight (usually dogs), normal bodyweight should be restored using an energy-dense, highly digestible diet, before placing it on a high-fibre diet. Diet is important to help glycaemic control, and water must always be available.

Cats and dogs have different dietary requirements. Dogs need a complex carbohydrate diet, including starch and dietary fibre, but avoiding simple sugars such as sucrose and restricted fat. This provides slow release of sugar into the blood over an extended period, preventing post-prandial glycaemic peaks.

Cats dietary requirements are relatively high in fat and protein content, and low in carbohydrates. Studies have shown, in a proportion of cats, that glycaemic control has improved and reduced insulin requirements, and such diets appear to reduce/eliminate the need for insulin therapy (Sparkes, 2009).

For DM patients to remain stable, a strict routine needs to be followed, including diet type and quantity, feeding times, insulin type and time of injection. The same amount of exercise every day should prevent any variation in insulin requirements (Figure 2). Exercise helps maintain glycaemic control by increasing the cells’ glucose use, and aid with weight loss. Excessive alterations to either food intake or exercise can upset the balance. It is much harder to control a cat’s exercise if it is mainly an outdoors animal, but indoor cats are becoming much more common, as is obesity. Keeping a record of exercise is an important part of a nurse’s role, as is the ability to notice trends and/or changes in a patient.

Nurses can devise a record card for hospitalised patients and owners. Examples are shown in Figure 3 and can be adapted to suit your veterinary practice’s needs. Intervet/Schering-Plough’s website shows a good example (www.vetsulin.com/PDF/Treatment_MonitSht.pdf).

It is important to take blood glucose levels before the patient is fed. Insulin should be given after feeding and three-quarters of the food has been eaten, to avoid a hypoglycaemic episode. Hospitalised patients don’t always eat well; in these situations, the vet will take into consideration the quantity eaten and the blood glucose level and may change the insulin dose.

When insulin is injected once daily, one-third of the daily food requirement should be fed in the morning, and the remaining two-thirds fed eight hours later, when the insulin should be at maximal
effect.

When insulin is injected twice daily, a dog’s daily food requirement should be equally divided into two – one fed before the morning insulin injection, and the other 12 hours later, with the second insulin injection.

It is advised to follow the same rule with cats, although ad-lib feeding is allowed as they like to pick at food. However, remember the quantity of food must not change (Simpson, 2004).

Studies have shown that if cats are fed strictly the correct diet, avoiding postprandial hyperglycaemia, the timing of meals is not critical in feline diabetics (Sparkes, 2009). In the morning, collect midstream urine and test it with a dipstick for the presence of ketones and glucose, and record the result. If ketones are present, advise owners to contact the surgery.

Owner compliance is very important; the patient’s routine must be maintained. Providing a record card to the owner will help him or her note changes in the pet, as well as making the patient’s history available to the surgery. The stabilised patient should be re-examined in one to two weeks, and then every three months, unless it deteriorates.

In cats, glucose curves obtained in the hospital are often difficult to interpret due to stress hyperglycaemia. Therefore, home glucose curves performed by the owner are becoming increasingly valuable. Low-volume, portable blood glucose monitors facilitate this. Blood samples obtained from an ear or paw prick have been used, but not all owners like to do this (Sparkes, 2009). Owners can regularly test urine for glycosuria and ketones using dipsticks. If ketones are found, the owner must consult the vet and not alter the insulin dose or dietary routine.

**Insulin handling**

Insulin must be stored upright (it sticks to the rubber lid) in the fridge and the bottle must be gently rotated – not shaken, as this will damage the particles and may make the insulin ineffective, before injecting. It is advised to change the injection site on a regular basis to prevent thickening of subcuticular sites, which is thought to reduce tissues’ability to absorb the treatment. The insulin vial must also be replaced on a regular basis to ensure viability of the product.

**Poor stabilisation**

If owner compliance is good, infection is the most common cause of destabilisation. Rule this out before changing insulin dosages. Otherwise, any increase in the dosage may result in hypoglycaemia once the infection is controlled. As previously explained, entire bitches must be spayed, and checks made for the presence of Cushing’s disease (dogs and cats) and acromegaly (cats).
Once these complications are controlled, insufficient insulin dose could be a cause. This will be seen on a glucose curve (repeated after 24 hours), where the blood glucose level never reaches the normal range (5-7mmol/l). If more than 2.2 units of insulin per kg are needed to control blood glucose concentrations, insulin effects may be antagonised, and will need to be investigated.

The duration of insulin action may be inadequate. This will be seen on the glucose curve, where blood glucose levels decrease initially but then rise, long before the next dose is required. Longer-acting insulin may be needed, or injections changed.

Too high a dose of insulin can cause hypoglycaemia. The body has a protective system to fight against it. It stimulates the release of hormones to antagonise insulin (such as glucagons, adrenalin and cortisol). These produce an effect called the Somogyi overswing, where hyperglycaemia is seen after a brief period of hypoglycaemia. The insulin dose needs to be altered (following the vet’s instructions) – probably reduced by half and then gradually increased, if needed, over a period of two to three weeks.

Other considerations should include poor owner compliance, such as not keeping to the pet’s routine, not feeding the correct diet, and poor insulin handling and injection technique.

**Insulin and syringe types**

The type of insulin may be described by the species of origin (such as bovine or porcine), type (such as lente, neutral, or protamine zinc – PZI), trade name (such as Caninsulin or Insuvet) and concentration (such as 40iu/ml or 100iu/ml). It is essential to use the appropriate syringe manufactured for the particular concentration of insulin. Different types of insulin syringes include U-100 insulin syringes, which are manufactured in 0.3ml, 0.5ml and 1ml capacity – these are orange-topped syringes (see below under the Insuvet [Pfizer] range). U-40 syringes are only available in 1ml capacity – these are red-topped syringes and are specifically manufactured for Caninsulin 40iu/ml (Intervet/ Schering-Plough).

**Licensed insulin types and syringes used**

- **Neutral/regular crystalline (eg, Insuvet neutral)**
  - There are no additions to delay absorption.
  - The onset of effect is immediately after IV administration, and 10 to 30 minutes after SC or IM administration.
  - The time to maximum effect is 30-120 minutes, with a duration of one to four hours.

- **Lente insulin (eg, Insuvet lente, Caninsulin)**
– Insulin (mixture of amorphous – short acting – and crystalline long acting) plus zinc.

– The onset of action is between 30 and 120 minutes.

– The biphasic peak action is at two to six hours and eight to 12 hours.

– The duration is reported to be between eight to 20 hours but, in my experience, it seldom seems to last more than 14 hours.

• Protamine zinc insulin (Insuvet PZI)

– It is made from a mixture of insulin, protamine (fish protein) and zinc to extend activity.

– The peak onset is between five to 20 hours, with a duration of eight to 24 hours.

Unlicensed insulin types include

• Insulin glargine (Lantus)

– 100iu/ml.

– It is a long-acting human insulin. It replaced asparagines with glycine and added two more arginines.

– It forms microprecipitate in tissues.

– It appears to act in a similar way to PZI.

UK licenced veterinary product ranges

• Insuvet range (Pfizer – bovine)

– 100iu/ml products use orange-topped syringes (U-100 syringe). The range comprises three different product lines: neutral, lente and PZI.

• Caninsulin (Intervet/Schering-Plough – porcine)

– 40iu/ml products use red-topped syringes (U-40 syringe).

– Lente.

Complications
Hypoglycaemia is a potentially serious complication of insulin therapy in the treatment of diabetes mellitus. Clinical signs are weakness, behaviour changes, altered mentation and ataxia, which can lead to collapse, seizures and brain damage if not addressed quickly. In the early stages of weakness or neurological changes, readily absorbed glucose products, such as honey, karo syrup and prescription diets suitable for canines and felines (eg, Hill’s a/d, Purina Veterinary diet CN [convalescence] and Royal Canin/Waltham Recovery). These diets are energy dense, and contain high concentrations of essential nutrients and highly digestible ingredients. They are soft, allowing them to be applied easily to the mucous membranes (use a spatula or syringe to avoid being bitten), and if there are signs of hypoglycaemia, the patient needs to be urgently seen by the vet. Glucose powder is only intended as a way to prevent a seizure prior to the vet seeing the patient – it will not last long. Intravenous glucose and supportive nursing care will be required. This would include a blood glucose base line, then hourly blood glucose measurements, with temperature, pulse and respiration rates monitored and recorded until the patient is stable.

**Diabetic ketoacidosis**

Ketones are formed during the breakdown of fats. Insulin is needed for the metabolism of ketones to carbon dioxide and water, as this prevents diabetic ketoacidosis (DKA). If a patient has been under physiological stress (such as an infection) or had pancreatitis, ketone production is increased and the rate is too rapid for ketones to be metabolised. This results in ketonuria, and metabolic acidosis can develop.

Clinical signs for ketoacidosis are polyuria/polydipsia, anorexia, vomiting and/or diarrhoea, depression, weakness or collapse, shock, poor body condition, an acetone smell on the patient’s breath and deeper, more rapid respiration, reflecting metabolic acidosis. Ascertaining when the patient last urinated is very important, as acute renal failure can develop in DKA (Skelly, 2007).

Blood glucose levels need to be measured immediately, monitored regularly and recorded to form a blood glucose curve; this may be every half hour initially, before reducing to hourly. Insulin is required to reduce hyperglycaemia by improving glucose use, thereby reducing ketone production; the dose will be decided by the vet.

Electrolytes, PCV, total protein, renal function, blood gas analysis (if possible) and urinalysis must all be tested at the start of an investigation to monitor and correct imbalances, thus improving intravascular volume and hydration status (Skelly, 2007).

Use an isotonic replacement fluid, either 0.9 per cent sodium chloride (NaCl) or Hartmann’s solution, depending on the electrolytes and blood gas results. Sodium chloride at 0.9 per cent is recommended initially, as total body sodium is often severely depleted. Potassium will need to be added, based on regular potassium measurements. Hartmann’s is controversial, as it contains potassium and this can be high in DKA. It is crucial that electrolytes are measured frequently, as potassium and phosphorous levels rapidly drop with insulin treatment.
Bibliography