Diabetic emergencies

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Physiology

The pancreas is an organ that is a mixed gland (endocrine and exocrine parts). The exocrine secretions enter the duodenum through the pancreatic duct. The endocrine secretions are formed in tissue found within the exocrine tissue known as the islets of Langerhans. The three hormones secreted from the islets of Langerhans are insulin, glucagon and somatostatin.

The insulin is secreted in response to high blood glucose levels in the body. Ultimately the insulin helps to lower the blood glucose by increasing the uptake of glucose into the cells and storing the excessive glucose as glycogen in the liver for later use. It is also responsible for breaking down fat and storing it as adipose tissue. Glucagon is secreted in response to low blood glucose levels. It helps to raise the blood glucose by converting the stored glycogen in the liver back to glucose. Somatostatin helps to regulate blood glucose levels in conjunction with insulin and glucagon as well as helps to decrease gut motility and secretion of digestive juices.

An insult to the pancreas occurs causing a degeneration in the islet cells. The insult could be from an acute or chronic onset of pancreatitis, neoplasm or may be idiopathic. The end result is that there is a reduction of the hormone insulin, the failure of the cells of the body to be able to respond to insulin and/or the increase of glucagon resulting in hyperglycaemia.

The lack of insulin in the body along with the rising levels of glucagon result in hyperglycaemia for an extended period of time within the body.

Hyperglycaemia

When blood glucose levels rise above 180mg/dl in dogs and above 280mg/dl in cats, the kidneys are no longer able to filter out all the glucose which results in glucose spilling into the urine. Glucose is therefore detectable on a urine test strip.

While blood glucose levels can reach above 600mg/dl, the only significant side effect to the extreme hyperglycaemia is dehydration of the tissue cells. The dehydration is likely because glucose does not diffuse easily through cellular pores which results in an increase in osmotic pressure in extracellular fluids causing fluids to shift out of the cells.

The loss of glucose in the urine leads to osmotic diuresis in the renal tubules causing a decrease in tubular reabsorption of fluid. This causes an increase in urine production, increase in thirst (due to the decrease of fluids being reabsorbed) and intra- and extracellular dehydration.

The chronic high glucose levels will lead to tissue injury of the blood vessels which causes the blood vessels not to function normally. The exact mechanism of why this occurs is not fully understood. When the blood vessels fail to function normally it leads to inadequate blood supply to the tissues. As the blood sugar increases pet can experience one of two types of emergencies: diabetic ketoacidosis (DKA) or hyperosmolar hyperglycaemic state (HHS).

DKA

Because there is a lack of insulin in the body, the body shifts from carbohydrate metabolism to fat metabolism. As the fat is metabolised for energy it produces pH falls below 7.0 coma or death may occur. In cases of severe metabolic acidosis (<7.0) the pet may experience Kussmaul respirations (slow, deep, and gasping respiration). The ketones build up in the body which eventually overwhelm the body. High numbers of ketones overwhelm the body's buffering system which leads to an increase in hydrogen ions (H+), a decrease in bicarbonate (HCO3-) and a decrease in blood pH. This leads to a metabolic acidosis. As the pH decreases and the dehydration worsens, the body will experience electrolyte abnormalities with sodium, potassium, phosphorous and magnesium.

Pets with DKA often present with symptoms including: polyuria, polydipsia, nausea, weakness, vomiting and diarrhoea. Other signs may include shock, tachypnoea, hypothermia, hypotension, and dehydration (dry oral membranes, decreased skin turgor, sunken eyes). About 10% of cats may exhibit diabetic neuropathy with a plantigrade stance (hocks touch the ground when the cat walks or stands). Approximately 40% of dogs may present with cataracts in one or both eyes. Hepatomegaly and secondary pancreatitis are common in both the dog and cat.

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HHS

While not common, HHS can occur in both the dog and cat and is sometimes referred to as ‘non-ketonic hyperglycaemia’. HHS is when the pet experiences profound hyperglycaemia (>600mg/dl), hyperosmolarity (>350mOsm/L), extreme dehydration, CNS depression and is absent of ketones and severe metabolic acidosis. Pets with HHS are more likely to have some underlying kidney or cardiovascular disease. HHS pets may also be non-insulin-dependent.

It's not fully understood why the pet does not experience ketonuria since the pathogenesis is similar to that of an uncomplicated diabetic. It is thought that there are likely some functioning cells that are still producing some insulin for the pet. The existence of some insulin prevents the formation of ketones. As the blood sugar rises the pet begins to experience severe signs of dehydration. As the dehydration continues the pet will start to experience azotaemia. The increase in osmolarity causes water to shift out of the cerebral neurons which results in obtundation.

Pets with HHS are often lethargic, severely dehydrated, depressed and weak. They may have gastrointestinal signs (anorexia, vomiting, diarrhoea) which may progress to neurological symptoms. They are often in shock and hypothermic. As it progresses the pet may experience a coma and/or death.

Diagnosing HHA and DKA

Diagnosis is made based on laboratory findings. A complete blood count (CBC), blood chemistry, electrolytes, urinalysis (sterile sample ideal) and blood gas should be performed. In both the dog and cat, a mild polycythaemia (due to dehydration) or leukocytosis may be noted.

A chemistry may reveal increased liver values, most commonly ALT and ALP. Increases in blood glucose level, kidney enzymes and cholesterol are often noted. Electrolyte derangements are more common in DKA and most commonly include hyponatremia, hypomagnesaemia, and hypokalaemia. If HHS is suspected serum osmolarity should be calculated. To calculate out osmolarity the following equation should be used: Serum Osmolarity = (2 x serum sodium

[mEq/L]) + (BUN [mg/dl]/2.8) + (glucose [mg/dl]/18). Normal serum osmolality is 290 to 310mOsm/kg. Neurologic symptoms have been documented in animals when osmolality exceeds 340mOsm/kg.

A urinalysis will always reveal glucosuria and, most likely, in the case of DKA, ketonuria. Protein, bacteria (due to secondary urinary tract infection) and/or blood may also be present in the urine. If DKA is suspected and ketones are not detected on a urine stick, testing plasma on a urine strip should be performed. It has been shown that plasma will detect ketones at times when urine may not.

Hypoglycemia

Hypoglycaemia is one of the most common side effects of insulin therapy in a diabetic. Clinical hypoglycaemic is defined as blood glucose levels less than 60mg/dl. Diabetics who experience a hypoglycaemic state do so because of one of two reasons: they were incorrectly dosed with insulin or are experiencing a change in insulin requirements. Cats frequently can experience ‘transient diabetes’ causing their insulin requirements to change. Approximately 20% of diabetic cats will experience transient diabetes. Changes in the pet's weight, appetite, diet, and activity level can results in the pet needing a lower insulin dose. Owners of new diabetics should be given written instructions on ways to avoid hypoglycaemic episodes (give insulin after ensuring pet has eaten, have family member double check dose, keep a check system on the refrigerator).

Hypoglycaemia mainly affects the nervous and musculoskeletal systems. Nervous tissue relies heavily on glucose as its primary energy source. While emergency signs resulting from hyperglycaemia take days if not weeks to occur, a pet experiencing hypoglycaemia is always an immediate emergency. Pets who experience hypoglycaemia will often become lethargic, weak, ataxic and appear ‘wobbly’. Signs can be seen in blood glucose levels between 50-60mg/dl. Hypoglycaemia stimulates appetite so pets may start acting hungry or eat things they normally may not. As the hypoglycaemia worsens (the blood sugar level continues to decline) the symptoms will worsen and can lead to seizures, coma, and death.

Treatment

Upon presentation of any emergency diabetic all emergent signs should be treated. Patients may present severely lethargic, shock, obtunded, seizuring or in a coma. All known diabetics should immediately have their blood glucose level tested to ensure the signs are not from hypoglycaemia. In general treatment should be focused on fluid therapy, insulin therapy if hyperglycaemic, electrolyte correction and treatment of any metabolic acidosis. It will take about 36-48 hours to normalise high glucose levels and pH levels and about 12-24 hours to normalise low glucose levels.

DKA, HHS and hypoglycaemic diabetics will present with signs and symptoms that warrant fluid therapy and medication administration (Valium, dextrose). Therefore, it is better if an intravenous (IV) catheter is placed first even if the patient is seizuring. A central line should be placed once the patient is a little more stable as this will allow for blood to be obtained and fluid therapy to begin almost simultaneously. In the case of a DKA patient, placement of a central line can usually occur first and is likely the only IV catheter placement needed. Placement of a central line will allow for central venous pressure (CVP) to be monitored, parental nutrition to be given and blood obtained for future lab work.

If the patient is experiencing HHS or is a DKA, baseline bloodwork should be obtained and fluid therapy should begin. Fluid therapy may be started before bloodwork is obtained if the patient is too critical and blood too difficult to obtain. The goal of fluid therapy is to reestablish

normal fluid balance. Just rehydrating the patient will aid in helping to decrease plasma glucose levels by helping to improve kidney function. The choice of fluids is ultimately the clinician's decision and should be based on electrolyte and acid-base status. Many clinicians will start with 0.9% NaCl to help with hyponatremia that most DKA experience. The current recommendations by the American Diabetes Association for patients with HHS is to use 0.9% NaCl in patients with a low serum sodium concentration and use 0.45% NaCl in patients with a normal or high serum sodium concentration. It is generally recommended to correct about 75% of the dehydration over the first 24 hours and the other 25% on the second day.

Potassium supplementation is usually added to the fluids of DKA patients. After 6-12 hours the electrolytes and blood gas should be rechecked in any HHS or DKA patient. Some clinicians will opt to change the fluids to a buffered solution with less sodium (LRS, P-lyte, Norm R) if the patient's sodium level is >140mmol/L.

Phosphate supplementation may be needed in a DKA patient due to a rapid decrease in levels within 12-24 hours after insulin administration begins. Phosphorus levels <1.5mg/dl can lead to anaemia, weakness, ataxia, and seizures. Phosphorus should never be used in conjunction with calcium containing fluids.

The use of bicarbonate therapy in DKA patients is controversial, but should be considered in patients with a pH <7.1 or a serum HCO3 <12mEq/L. Most of the time acidosis improves once perfusion and glucose levels are normalised. Using a buffered solution (LRS, Norm-R) also aids in the correction of any acidosis. Administration of bicarbonate can result in coronary acidosis, a paradoxical CNS acidosis and therefore sudden death. If the patient is not responding and still in a critical acidotic state, then bicarbonate therapy can be initiated. Bicarbonate must be administered slow (over 2-6 hours) to avoid overwhelming the blood-brain barrier with rising levels of PCO2. The use of bicarbonate is not recommended in HHS patients and not needed in hypoglycaemic patients.

If an owner calls stating their pet is a known diabetic who was overdosed on insulin or who is exhibiting signs of hypoglycaemia (seizure, ataxia) they should be instructed to put karo syrup, honey or even raw sugar on the gums and then drive to the veterinary hospital immediately. Upon arrival, if the pet is experiencing hypoglycaemia an initial slow IV bolus of 50% dextrose (0.5g/kg diluted with sterile saline or sterile water 1:4 or 1:2 ratio) should be administered. The pet should then be placed on a CRI of 5% dextrose solution.

Blood sugars will be constantly checked every couple of hours. Hypoglycaemic pets should slowly be weaned off the dextrose CRI over a 12- to 24-hour period once they have high or normal blood glucose levels. The cause of the hypoglycaemia must be investigated so that further hypoglycaemic episodes do not occur. This may be a decrease in insulin, removal of insulin all together or more careful medical management at home.

Insulin therapy

Regular insulin is always chosen for the initial treatment of DKA and HHS because it offers a quick onset of action and a short duration of effect. There are two routes regular insulin can be given to patients: IV or IM. If given IV, it is typically given at a constant rate infusion (CRI). Intramuscular doses are given intermittently, usually every 4-6 hours with blood glucose checks occurring every two hours. If a DKA patient is on a CRI of insulin, a dextrose CRI should be added once glucose levels reach between 150-300mg/dl. Adding in dextrose will help with the further breakdown of the remaining ketones and help to resolve acidosis of a patient with DKA.

The goal of insulin therapy in a DKA is to drive blood glucose levels between 150-300mg/dl. In the case of a pet with HHS, it is recommended that blood glucose concentration remain at 250-300mg/dl until hyperosmolarity is corrected. DKA patients are then started on subcutaneous regular insulin until anorexia and ketosis has resolved. Pets are then sent home on either intermediate or long-acting maintenance insulin.

Nursing Care

Nursing care of diabetic emergencies can be intense. The primary nursing care of a diabetic emergency patient revolves around monitoring the patient's response to the treatment. Physical exam parameters and blood pressure should be checked every 2-6 hours depending on how critical the pet is. Pets should be weighed twice a day to monitor rehydration. Since these pets are often on high rates of fluids, they should be monitored closely for signs of fluid overload (respiratory rate/effort, auscultation of lung sounds).

A urine catheter should be considered in down pets to aid in keeping them clean and to be able to quantify urine output. Non-absorbent litter or obtaining a free catch can be done to obtain accurate amounts of urine. Urine should be checked every 6-12 hours for ketones so collection of urine is important for a variety of reasons. Ketones may persist up to five days in the urine even after a resolution of the ketosis state.

Measuring CVP will help determine how much fluid can be administered to a patient without causing fluid overload. Depending on the literature normal CVP measurements vary, but most will agree it is somewhere between 1-10cm H2O. Monitoring CVP is particular useful in HHS and DKA patients.

It is important that patients have their blood pressure monitored minimally every 4-6 hours. If the mean arterial pressure (MAP) falls below 60mmHg, the kidneys and other organs are not appropriately perfused putting them at risk for organ failure. Normalisation of blood pressure, defined by a of MAP of 80-120mmHg or systolic between 110-160mmHg, is goal.

Diabetic emergency patients require frequent blood draws, and it is imperative that if the patient does not have a central line, the integrity of the veins be kept in good health. Having one ready to go and placing a pressure bandage on for every venipuncture is imperative.

If the patient is recumbent, the pet will need to be turned and kept dry and free of bed sores and have passive range of motion must be performed every four to six hours.

Lastly it is important that patients be given appropriate nutrition. Malnutrition can delay healing and cause large fluctuations in glucose/insulin regulation. Feeding tubes, force feedings or parental nutrition should be considered in patients who are unwilling to eat.

At time of discharge clients need to have a discussion on how to care for their diabetic animal. Handouts and one-on-one demonstrations on insulin handling and administration is key to client compliance.

Conclusion

As a veterinary technician you will likely encounter an emergency diabetic pet at some point. Being able to communicate with the owner quickly and effectively will allow for faster treatment of the pet. Being able to monitor the patient and notice subtle changes will help the patient receive the appropriate course of treatment and allow for a faster recovery.

References available upon request.