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DIABETES KETOACIDOSE: A PRACTICAL APPROACH

Diabetic ketoacidosis (DKA) has always been a challenging disease. During the past decade the treatment and monitoring of these cases has changed, became more intense, resulting in an improved prognosis! It is therefore a pleasure to share the benefits of constant rate infusions (CRI) of insulin, regular electrolyte monitoring and investigation of underlying causes.

The diagnosis of diabetes mellitus (DM) is straightforward, requiring the identification of hyperglycaemia and glycosuria. However, be aware that alfa2-agonists such as medetomidine cause a rise in glucose concentrations, which may even lead to glucosuria, and therefore to a false diagnosis of DM in a sedated cat. Moreover, stress may also cause hyperglycemia, especially in cats, which when prolonged will also result in significant glucosuria. Fructosamine levels, which reflect long term blood glucose levels are especially helpful in these cases. Fructosamine levels are influenced by protein concentrations and metabolism though, and thus hyper- or hypoproteinaemia and hyper- or hypothyrodism will modify concentrations. The diagnosis of DKA requires the additional identification of ketone bodies in the urine, and a decreased venous blood pH. DKA patients usually have a history of: unidentified DM, ill-treated DM or DM with a complicating factor such as a pancreatitis. Therefore, every DKA-patient requires a further work-up to rule out an underlying pancreatitis, or other inflammatory, infectious or neoplastic process.

The initial treatment of DKA patients aims at establishing normovolemia and normohydration, whilst normalizing pH, electrolyte levels and only slowly (decreasing glycemia by 50 to 100mg/dL/h) re-establishing 'acceptable blood glucose levels'. Fluid resuscitation provokes a moderate drop in glucose levels, and normalizes blood pH. Potassium levels can be whatever at presentation: the lack of insulin and hyperglycaemia impede movement of potassium into the cells, resulting in intracellular potassium depletion. Moreover, osmotic diuresis increases potassium excretion, resulting in hypokalemia. However, severely dehydrated patients have decreased potassium excretion which can cause extracellular hyperkalaemia. The veterinarian must know that insulin will rapidly shift potassium into the intracellular compartment, which can rapidly result in hypokalaemia. Similarly, DKA patients not seldomly develop hypophosphatemia, which can induce haemolytic anaemia amongst other signs. DKA patients require intravenous access for fluid administration, electrolyte supplementation and ideally continuous low-dose insulin administration. Electrolyte and glycaemic control require regular blood samples (glucose levels could be checked using glucometers via peripheral samples from footpads or ears), and therefore central venous lines in these patients facilitate the life of both the patient and veterinarian in charge.

Insulin is ideally administered via continuous rate infusions (CRI) of short acting insulin, or alternatively via intramuscular and subcutaneous injections. Intravenous administration ensures bioavailability, opposed to subcutaneous and intramuscular injections. Moreover, insulin resistance can disappear in DKA patients when volemia and glucose levels improve, and therefore a short acting molecule is preferrable. As stated previously, glucose levels should slowly decrease (to avoid osmotic shifts between the blood and the neurons) by 50-100mg/dL/h. The goal is to obtain glucose levels around 10 to 8 mmol/L or 180-144mg/dL, which is low enough to avoid osmotic diuresis, yet high enough to avoid hypoglycaemia. When glycemia decreases underneath 10mmol/L (180mg/dL), a constant rate infusion of a 5% glucose solution at 2mL/kg/h is added, to preventing hypoglycaemic events.

Central venous lines allow to obtain blood samples more easily, and monitor central venous pressure to guide fluid therapy. Electrolytes (and blood gasses) must be assessed every 6 to 8 hours initially, together with glucose levels to titrate the insulin CRI. A urinary tube to monitor output and calculate 'ins and outs', and offering rapid access to urine samples is often indicated (considered against the risk for urinary tract infections).

Obviously these patients also require additional supportive or curative treatment for any underlying disease (opioids for abdominal pain in pancreatitis, antiemetics, antacids for gastric ulceration etcetera). During the initial days, patients must be stimulated to eat, or a nutritional tube should be placed. CRI's can be switched to injections of regular

long acting insulin as soon as the patient is stable, glycaemic control is achieved and the patient is eating (or fed via a tube) and has stopped vomiting.

In summary: DKA patients are challenging cases, but have a fair prognosis when the owners and veterinarian are motivated. However, to obtain good long-term objectives, client education is imperative. Finally don't forget: glucose and fructosamine levels don't have to be perfect! Our pets rarely live long enough to develop necrotic lesions at their extremities, and the development of cataracts is likely, even despite excellent glycaemic control. Owners and pets will often be happy when osmotic diuresis has stopped, weight loss has ended and appetite has normalized…!