SAIM

Case Log #11

April 9, 2023

“Wally” Herrman - 459404

Wally, a 10 year old, male neutered, domestic shorthair feline presented for anorexia, lethargy, polyuria, polydipsia, and urinating outside of the litter box. Physical examination showed a generalized normal outer appearance consisting of a normal coat and a 5/9 body condition score. Mentation was appropriate, quiet, alert, and responsive. Abnormal findings consisted of a dehydration status of 5-7%, mild painful abdomen on palpation (pain score of 1/4, using the Colorado State University Feline Acute Pain Scale), and light pink and tacky mucous membranes. Initial vital signs included a BW of 4.73 kg, heart rate of 180 bpm, respiratory rate of 32 breaths per minute with no respiratory effort, a temperature of 99.5 degrees F, and a non-invasive doppler BP reading of 125 millimeters of mercury (mmHg), all within normal parameters. No heart murmur or abnormal lung sounds were noted during auscultation. Wally had no previous medical history and was not taking any medications prior to presentation.

Initial diagnostics included a CBC, serum biochemistry profile, venous blood gas analysis, and urinalysis. CBC results revealed neutrophilia of 12.46 thousand (K) per microliter (uL) (reference range: 2.30K/uL - 10.29K/uL) and monocytosis of 1.27K/uL (reference range: 0.05K/uL - 0.67K/uL). Serum chemistries showed hyperglycemia with a blood glucose (BG) value of 429mg/dL (reference range: 71mg/dL - 159mg/dL), increased globulin level of 5.5g/dL (reference range: 2.8g/dL - 5.1g/dL), increased ALT of 235u/L (reference range: 12u/L - 130u/L), increased ALKP of 117u/L (reference range: 14u/L - 111u/L), elevated T.Bili at 1.9mg/dL (reference range: 0.0mg/dL - 0.9mg/dL), elevated cholesterol level of 365mg/dL (reference range: 65mg/dL - 225mg/dL), and decreased amylase level of 421u/L (reference range: 500u/L - 1500u/L).

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Electrolyte abnormalities from chemistry analyzer included an increased sodium (Na) value of 172mmol/L (reference range: 150mmol/L - 165mmol/L) and a decreased potassium (K+) value of 3.1mmol/L (reference range: 3.5mmol/L - 5.8mmol/L). Venous blood gas analysis revealed a low blood pH of 7.143 (reference range: 7.230 - 7.423), a decreased bicarbonate (HCO3) of 9.4mmol/L (reference range: 16.8mmol/L - 24.0mmol/L), and a low base excess (BE) of -18.1mmol/L (reference range: -9.5mmol/L - -0.7mmol/L) consistent with metabolic acidosis.

The applicant performed an ultrasound guided cystocentesis to obtain a sterile urine sample to send for urinalysis and culture to an outside reference laboratory. A small urine sample was analyzed in the hospital to evaluate glucose and ketone levels by the applicant. Urine dipstick results revealed a mild amount of both glucose and ketones in Wally’s urine sample. A positive result of ketonuria and glucosuria is an outcome of prolonged hyperglycemia and conversion to ketosis.

The problem list for Wally consisted of inappetence, inappropriate eliminations, weight loss, neutrophilia, monocytosis, hyperglobulinemia, hypernatremia, elevated liver enzymes, hypokalemia, mild ketonuria, hypercholesterolemia, metabolic acidosis, and hyperglycemia. Prognosis at this time was guarded due to all the abnormalities in Wally’s initial diagnostics, and a positive outcome is dependent on response to treatment. Differential diagnosis included diabetic ketoacidosis, pancreatitis, hepatic lipidosis, and urinary tract infection.

Wally was hospitalized for supportive care including IV fluids, an insulin CRI, serial blood glucose monitoring, serial electrolyte measurements and blood gas analysis, as well as a nutritional support regimen.

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The applicant placed a 20 gauge IV catheter into the left cephalic vein for medication and fluid administration. Due to the need for frequent blood sampling, the applicant found it beneficial to place a multi lumen central catheter. The applicant administered a butorphanol dose of 0.2 mg/kg, 0.95 mg IV to provide sedation and mild pain relief for placement of the catheter. The applicant prepared the right ventral neck of the patient by clipping the hair and aseptically preparing the venipuncture site. The applicant placed a 20 gauge over-the-needle catheter as an introducer, then proceeded to use a modified Seldinger technique to place a 5.5 French x 13 cm triple lumen catheter. The applicant confirmed patency of all ports, and sutured the catheter in place. The applicant performed a lateral thoracic radiograph to verify placement of the catheter just proximal to the right atrium.

Initial treatment for Wally included IV fluid therapy of a balanced isotonic crystalloid solution at a constant rate of 4.23mL/kg/hr, 20mL/hr. The applicant administered maropitant 1 mg/kg, 4.73 mg IV q 24 hours to help with nausea and initiate gastrointestinal support. DACVIM (SAIM) ordered ampicillin-sulbactam and the applicant calculated and administered a dose of 30 mg/kg, 141.9 mg IV q 8 hours to treat for potential urinary tract infection, while a urine culture was pending. An insulin CRI order of 2.22 international units (IU)/kg was created and the applicant prepared a total dose of 10.5 IU of short-acting insulin diluted into 250mL of sterile saline to be given at a constant rate of 10mL/hr via a dedicated catheter. The applicant primed the IV fluid line with 50mL prior to administration, due to the potential mechanism of insulin binding to plastic, ensuring an accurate insulin dose was administered to the patient. The total quantity of fluids per hour to be given to the patient was 20mL. When taking the insulin CRI into consideration, this prompted the applicant to decrease the crystalloid solution to 10mL/hr. K+

supplementation was initiated to correct the hypokalemia on initial blood work, and in anticipation of further decline of blood K+, due to insulin causing movement of K+ into the cells. The applicant calculated and added a potassium chloride (KCl) dose of 0.126mEq/kg/hr, 60mEq/L into the patients IV fluids. Nursing care treatment considered by the applicant included monitoring hydration status, including monitoring for frequent urination, as is expected with diabetic patients.

On day 2 of hospitalization, an abdominal ultrasound was performed to evaluate Wally’s liver, pancreas, GI tract, and to evaluate any potential incidental findings. The applicant assisted DACVIM (SAIM) and restrained Wally in ventrodorsal recumbency for ultrasound imaging. Abdominal ultrasound revealed a hyperechoic, enlarged liver, a thickened duodenum, hyperechoic mesentery surrounding the pancreas, and prominent abdominal and mesenteric lymphadenopathy. The hepatomegaly was consistent with diabetes mellitus and the ultrasound provided a diagnosis of pancreatitis. Further diagnostics included thoracic radiographs, repeat venous blood gas measurements every 6 hours, and blood glucose measurements via a glucometer every 2 hours. Thoracic films were unremarkable. BG values remained between 200mg/dL - 410mg/dL (reference range: 70mg/dL - 120 mg/dL). Wally’s repeat venous blood gas analysis showed persistent and worsening hypokalemia of 2.24mmol/L (reference range: 3.19mmol/L - 4.76mmol/L), an improved sodium level of 159.7mmol/L (reference range: 149.8mmol/L - 155.7mmol/L), an improved blood pH of 7.23, and hypocalcemia of 0.98mmol/L (reference range: 1.19mmol/L - 1.35mmol/L). Due to the refractory K+, an additional blood test was ordered to evaluate Wally’s magnesium blood level. Magnesium result was 1.24mg/dL (reference range: 1.50mg/dL - 3.00mg/dL). Hypomagnesemia is known to cause persistent

hypokalemia and prevent adequate response to K+ supplementation. To help correct the low magnesium level, a magnesium CRI was initiated. The applicant calculated and administered a dose of 2 mEq/kg/day, 9.46 mEq/day IV of magnesium supplementation over 8 hours. Additional therapeutics added included the administration of pantoprazole 1 mg/kg, 4.73 mg IV q 12 hours to reduce gastric acidity, buprenorphine 0.02 mg/kg, 0.09 mg IV q 8 hours to relieve abdominal pain from pancreatitis, and an increase to the ampicillin-sulbactam dose to 50 mg/kg, 236.5 mg IV q 8 hours for a more robust coverage of bacterial infection.

Wally remained anorexic warranting placement of a nasogastric (NG) tube and initiation of nutritional support. The applicant administered an alfaxalone dose of 1 mg/kg IV to provide sedation for feeding tube placement. The applicant placed a 6 French NG tube in the left nostril at the 35cm mark, and sutured it in place. A right lateral radiograph was performed to confirm appropriate placement in the stomach by DACVIM (SAIM). A nutrition plan was created using the RER formula involving raising Wally’s body weight to the power of 0.75 and multiplying it by 70, which resulted in a daily caloric requirement of 224.5 Kcal/day. Feeding orders commenced at one-third of the daily requirement equaling 74.8 Kcal/day, rounded to 75 Kcal/day. A CRI of a renal liquid diet (0.9 Kcal/mL concentration) was calculated and administered by the applicant at a rate of 3.5mL/hr. The feeding plan included raising the daily RER by ⅓ until 100% RER was attained. Nursing considerations during nutritional CRI was to monitor for nausea, vomiting, or regurgitation. Wally tolerated CRI feeding well during hospitalization.

On day 3 of hospitalization, Wally’s electrolyte derangements continued. A low phosphorus level of 1.0mg/dL (reference range: 3.1mg/dL - 7.5mg/dL) was obtained overnight.

Potassium phosphate (KPhos) supplementation was started at a dose of 0.01 mmol/kg/hr, 4.73 mmo/L and added to his crystalloid solution. Reevaluation of magnesium post CRI showed a mildly elevated blood level of 4.41mg/dL. Recheck venous blood gas and electrolyte analysis revealed a mildly elevated blood pH of 7.485, elevated bicarbonate of 32.6mmol/L, elevated base excess of 8.2mmol/L, reduced sodium level of 141.3mmol/L, persistently low potassium level of 2.11mmol/L, ionized calcium level of 0.98mmol/L, and reduced chloride level of 91mmol/L (reference range: 113mmol/L - 121mmol/L). BG values remained between 239mg/dL - 461mg/dL (reference range 70mg/dL - 120mg/dL). Due to the persistent hypokalemia, a high supplement dose of K+ (K max) was calculated and administered by the applicant per DACVIM (SAIM) orders. A Kmax dose of 0.7 mEq/kg/hr, 66.22 mEq/20 hours was diluted with 100mL of sterile saline for administration at a rate of 5mL/hr, equaling a total dose of 3.31 mEq/hr. Further IV fluid therapy adjustments included a decrease in KPhos supplementation to a lower dose of 0.006 mmol/kg/hr, 0.028mmol/hr, a balanced crystalloid solution at a rate of 2mL/hr, and an insulin CRI containing 4.4 IU/kg, 20.8 IU of short-acting insulin diluted into 250mL of sterile saline to be given at a constant rate of 5mL/hr. Fluid therapy goal was a total of 12mL/hr including insulin CRI, electrolyte supplement solution, and crystalloid solution. Insulin CRI dose was doubled to permit fluid rate decrease. A single dose of spironolactone 1.32 mg/kg, 6.25 mg PO was given due to its potassium sparing effect. Nursing considerations during this point of therapy included intense monitoring of Wally’s cardiovascular system. The applicant began continuous telemetry monitoring to observe for any arrhythmias, including flattening/loss of P waves and tall T waves. Wally maintained a normal sinus rhythm during hospitalization. The applicant offered various types of diet intermittently to gauge a

change in appetite, however, at this time, Wally had no interest in eating. No change in his nutritional CRI was performed at this time.

Day 4 of hospitalization showed a drastic improvement in Wally’s response to treatment. BW improved to 4.92 kg. Overnight, an upward trend in K+ levels warranted a decrease in supplementation. Venous blood gas and electrolyte analysis revealed a blood pH of 7.433 (reference range: 7.230 - 7.423), mild elevation of bicarbonate of 29.5mmol/L (reference range: 16.8mmol/L - 24.0mmol/L), mild elevation of base excess of 4.6mmol/L (reference range: -9.5mmol/L - -0.7mmol/L), low normal sodium level of 149.0mmol/L (reference range: 149.8mmol/L - 155.7mmol/L), corrected and mildly elevated potassium level of 5.4mmol/L (reference range: 3.19mmol/L - 4.76mmol/L), corrected ionized calcium level of 1.25mmol/L (reference range: 1.19mmol/L - 1.35mmol/L), and low normal chloride level of 105mmol/L (reference range: 113mmol/L - 121mmol/L). Recheck phosphorus level showed a resolution of deficiency with a value of 3.4mg/dL (reference range: 3.1mg/dL - 7.5mg/dL), prompting discontinuation of all electrolyte supplementation. Urine culture results from the outside reference laboratory returned negative for growth. BG values remained between 223mg/dL - 330mg/dL (reference range 70mg/dL - 120mg/dL). Wally’s appetite improved and began to eat a gastrointestinal diet on his own. With Wally’s appetite improved, insulin CRI was discontinued and therapy with a long-acting insulin was started. The applicant administered an initial insulin dose of 0.2 IU/kg, 1 IU SQ q 12 hours (calculated with new current weight). BG values post insulin injection remained above 600mg/dL throughout the night, with a nadir of 602mg/dL. On the following day (day 5 of hospitalization), an increased insulin dose of 0.4 IU/kg, 2 IU SQ was started.

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Wally was discharged after 5 days in the hospital for a diagnosis of diabetic ketoacidosis (DKA) and pancreatitis. Intense monitoring and supportive care was necessary due to Wally’s severe electrolyte imbalances. With the return of his appetite and electrolyte correction, Wally was clear for discharge to continue at-home insulin therapy. The absence of pain on abdominal palpation also gave evidence of the pancreatitis improving and potentially resolved. However, serial reevaluations would be necessary to monitor glycemic control and adjust therapy as needed. Discharges reviewed with the owner by the applicant, included a diabetes overview, insulin instructions, and a SC injection demonstration. The applicant thoroughly explained the symptoms of hypoglycemia and hyperglycemia to the owner, to promote careful and successful monitoring at home.

Diabetes mellitus (DM) in felines compares to type 2 diabetes in humans and is usually due to insulin resistance, which is the body’s inability to respond to insulin, secondary to desensitization of insulin receptors on cells. Causes of feline DM are usually due to islet cell amyloidosis, obesity, and chronic pancreatitis. Insulin and amylin are secreted together from beta cells in the Islet of Langerhans of the pancreas. Felines with insulin resistance experience an increase in amylin secretion, and the overproduction of this progresses to diabetes, due to amylin converting to amyloid and having a cytotoxic effect on islet cells (Bruyette & Eiler 2013). Diabetic cats are about six times more resistant to insulin than normal. Weight gain plays a major role in making cats resistant to insulin. An increase in weight by just one kg causes a 30% decrease in insulin sensitivity (Ettinger et al., 2017). Pancreatitis may result in loss of pancreatic islets and is diagnosed concurrently with DM in as many as 60% of felines. Although pancreatitis does not commonly cause DM, it can play a role in beta cell loss and minimize the

likelihood of remission (Ettinger et al., 2017). Insulin allows glucose, absorbed and manufactured by the body, to get into body cells for utilization. Without insulin, glucose cannot enter the cells, and the body experiences a state of starvation. Glucose builds up in the bloodstream and as a result, will also spill into the urine. Symptoms of high glucose level in the blood involve excessive drinking and urination, weight loss, and polyphagia. Wally was experiencing a few of these symptoms, however, with the combination of pancreatitis, Wally’s appetite was affected leading to anorexia at home.

Due to the inability to utilize glucose, the body resorts to ketosis and the development of ketone bodies for energy (ATP) production. The production of ketones leads to diabetic ketoacidosis, a life threatening emergency, which Wally presented with. Cellular starvation of glucose leads to oxidation of free fatty acids (FFAs), which leads to hepatic production of ketone bodies as an alternate energy source for the liver and peripheral tissues. Production of ketones are also triggered by the release of glucose counter-regulatory hormones, such as epinephrine, norepinephrine, cortisol, glucagon, and growth hormone (Rudloff, 2017). A diagnosis of diabetic ketoacidosis is supported by the presence of ketones and metabolic acidosis in laboratory results.

The goal of therapy for DKA patients is to correct dehydration status, supplement electrolytes, while trying to obtain glycemic control with insulin administration, and cease ketogenesis. Fluid therapy with a balanced crystalloid solution will aid with the patient’s hydration status, correct hypovolemia, improve glomerular filtration, resolve hypernatremia, and supplement potassium and phosphorus (Thomovsky, 2017). In Wally’s specific case, extreme electrolyte imbalances warranted further electrolyte supplementation throughout his

hospitalization. Wally’s continued care is currently being monitored by the primary veterinarian. Owner was advised to reach out to us if any further care was needed.

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