WHAT'S YOUR DIAGNOSIS?

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CASE:

Sammy, a 15 year old male neutered domestic short-hair cat, presents to you for lethargy and weakness. He has been diagnosed with hypertrophic cardiomyopathy and renal insufficiency in the past. He is not currently receiving any medication. Sammy also has a history of weight loss over the past few months. He has had a good appetite until the last day or so. He has been polyuric and polydipsic for the last few days. There is no history of vomiting. He has not urinated in the last 12 hours.

On physical examination, Sammy is mentally dull. His mucus membranes are tacky and pale and his capillary refill time cannot be determined. Sammy is 8 to 10% dehydrated. His heart rate is 170 bpm and the pulses are weak but palpable. The respiratory rate is 24 brpm. The rectal temperature is 38.0 °C. Thoracic auscultation and abdominal palpation are unremarkable. Sammy's body condition is 2/5 with evidence of weight loss and muscle wasting. The Doppler blood pressure is 90 mmHg.

Quick assessment tests obtained from a venous blood sample show the following:

- PCV 18% (30 – 50%)
- Total solids 68 g/L (55 – 75g/L)
- pH 7.160 (7.25 – 7.35)
- pCO_2 40.7 mmHg (28 – 50)
- Na^+ 177.5 mmol/L (150.6 – 155.8)
- K^+ 5.78 mmol/L (3.17 – 5.38)
- Cl^- 149 mmol/L (120 – 128)
- Glucose 30 mmol/L (3.9 – 6.4)
- Lactate 1.0 mmol/L (0.5 – 2.5)
- Base Excess -13.1 mmol/L (-10.5 – -5.4)
- HCO_3^- 13.9 mmol/L (15.6 – 21.4)
- Anion Gap 20.3 mmol/L (14 – 18)
- A urine sample is obtained by cystocentesis and shows 3+ glucosuria and a urine specific gravity of 1.014. The urine is negative for ketones.

What is your diagnosis?

ANSWER:

Based on the physical exam findings, Sammy is markedly dehydrated and neurologically inappropriate. While his blood volume is likely diminished, his perfusion is adequate. The signalment, history, physical exam and bloodwork (a metabolic acidosis with an elevated anion gap) findings are consistent with diabetic ketoacidosis; however Sammy does not have ketonuria. Calculation of effective osmolality [$2(Na^+ + K^+)$ + glucose] reveals an effective serum osmolality of 396 mOsm/kg (normal range in cats: 290 – 320 mOsm/kg*). This finding, in conjunction with the hyperglycemia, gives us the diagnosis of 'Hyperglycemic Hyperosmolar Syndrome' (HHS), also known as 'Hyperglycemic Hyperosmolar Non-ketotic Syndrome'.

HHS results from a combination of relative or absolute lack of insulin, presence of counter-regulatory hormones, a decrease in glomerular filtration rate, and presence of concurrent disease. Counter-regulatory hormones that antagonize the effects of insulin include glucagon, corticosteroids, epinephrine and growth hormone. Serum levels of these hormones increase with the stress of concurrent disease. Fluid loss secondary to osmotic diuresis occurs with glucosuria. Fluid losses can also occur through vomiting, diarrhea, and decreased water intake. Subsequent dehydration results in hypovolemia and a reduction in glomerular filtration rate (GFR). The decrease in GFR will reduce glucose excretion and cause hyperglycemia to worsen. Concurrent diseases such as renal failure and congestive heart failure also contribute to decreases in GFR. Infection, neoplasia and other endocrinopathies perpetuate production of counter-regulatory hormones.

With HHS, insulin production may be adequate to prevent ketones from forming; however, the marked increase in osmolality associated with hyperglycemia and dehydration will cause neurologic signs associated with cerebral dehydration.
Cases like Sammy require aggressive monitoring as abrupt changes in serum osmolality can cause cerebral edema. Serum osmolality should not be decreased by more than 3 mOsm/kg/hr. Additional complicating factors in this case include Sammy's cardiac and renal insufficiency.

**Note: Units for serum osmolality reflect mOsm per kilogram of serum, not per kilogram of body weight.*