Small Animal Internal Medicine
Case Log 1
Date of Treatment: 10/05/22
Patient ID: 449682

Case Report 3

History

A 15 year and 3 month old, male neutered, feline, Ragdoll presented to the applicant's facility (received by emergency services) on October 4th, 2022 for further evaluation of diabetes mellitus (DM) and hyporexia noted over a two day period. DM was initially diagnosed in 2021 by their rDVM (managed with protamine zinc recombinant human insulin [PZI] 0.63 units/kg [2.5 units] SC BID) and multiple reported incidences of diabetic ketoacidosis (DKA) had occurred post DM diagnosis. Upon presentation, the patient's mentation was dull with 5-8% dehydration noted on physical examination (PE) (see **Appendix A**). CBC and venous blood gas (VBG) at that time were within normal limits, chemistry revealed hyperglycemia (634 mg/dL [N=71-159 mg/dL]), serum ketone analysis revealed 15 mg/dL (small) ketone presence (N= 0 mg/dL) (see **Appendix B**), and thoracic radiographs showed no significant findings (see **Appendix C**). Diagnostics were suggestive of diabetic ketosis and the patient was hospitalized with the following medical therapy: a balanced crystalloid solution with potassium chloride 20 milliequivalents per liter (mEq/L) at 5.06 mL/kg/hr (20 mL/hr) IV, maropitant 1 mg/kg (3.95 mg) IV SID, pantoprazole 1 mg/kg (3.95 mg) IV BID, short-acting regular insulin human injection 0.126 units/kg (0.5 units) IM q 2 hr PRN, and venous blood glucose (BG) monitoring q 2 hr via a veterinary glucometer calibrated to feline species (see **Appendix D**).

Patient Status on Presentation

After 12 hours of hospitalization and supportive care, the patient was transferred to the applicant's service on October 5th, 2022. Upon transfer, the patient was quiet, alert, and

responsive with a weight of 3.96 kg. Vital parameters obtained at that time revealed prehypertension (156 mmHg on indirect arterial Doppler blood pressure), normothermia (101.1 degrees ℉), a heart rate of 190 bpm with a pulse rate of 200, a respiratory rate of 32 with no noted respiratory effort, a capillary refill time (CRT) of <2 seconds, and light pink mucous membranes. The applicant performed a PE (see **Appendix A**), revealing no murmurs or arrhythmias on auscultation, clear bronchovesicular sounds bilaterally, pain elicited on abdominal palpation, and a body condition score of 4/9 (based on The World Small Animal Veterinary Association Global Nutrition Committee). The patient was well-hydrated at this time based on skin turgor, moist mucous membranes, and a CRT within normal limits. The applicant then performed a pain assessment and graded a 2/4 with the Colorado State University feline pain scale based on quiet mentation, hunched posture while resting, and an aggressive response elicited on abdominal palpation, which prompted the applicant to advocate for analgesic therapy. In addition to the previously diagnosed DM and newly diagnosed diabetic ketosis on presentation to the hospital, the patient clinically was suffering from hyporexia and abdominal pain based on PE.

Veterinarian's Differential Diagnosis

The veterinarian's differential diagnosis list for the elicited abdominal pain response included pancreatitis vs. an underlying enteropathy vs. other. With pancreatitis being highly suspected at that time, it was likely that insulin resistance secondary to pancreatitis had induced diabetic ketosis in this patient or that poor diabetic control and subsequent hyperglycemia had induced pancreatic inflammation.

Veterinarian's Initial Assessment of Prognosis

The veterinarian's initial assessment of prognosis in this patient was good. The absence of acidosis and present mild ketosis in this patient was a positive prognostic indicator in comparison to more severely unregulated DM. However, concurrent pancreatitis increases the complexities of diabetic management.

Interventions

Given the elicited pain response on abdominal palpation, an abdominal ultrasound (AUS) was performed for further evaluation, revealing scant peritoneal fluid and a hypoechoic and irregularly margined pancreas consistent with pancreatitis (see **Appendix C**). A urine sample was then obtained via ultrasound-guided cystocentesis using a 25 gauge 1.5 in needle without complication for urinalysis and culture. The patient's temperament was monitored during AUS as well as cystocentesis and mild discomfort was noted during imaging. A multi-modal analgesic plan was then proposed by the applicant to include gabapentin (a gabapentinoid and alpha-2-delta-1 subunit calcium channel blocker) 1.9 mg/kg (7.5 mg) PO BID PRN for its anti-hyperalgesic effects and buprenorphine (a synthetic opioid and partial mu agonist and mu antagonist) 0.01 mg/kg (0.04 mg) IV QID PRN for mild to moderate analgesia, which were administered with DACVIM approval. The applicant then placed a peripherally inserted central catheter (PICC) to facilitate continued q 2 hr BG monitoring. The right medial saphenous was clipped and aseptically prepared with alcohol and 4% chlorhexidine. Applicant, with sterile technique, placed a 3 French PICC in the right medial saphenous vein using a through-the-needle (peel-away) 19 gauge catheter after measuring and trimming the central catheter to reach the caudal vena cava, just cranial to the level of the posterior superior iliac spine. The catheter was secured with 3-0 synthetic nonabsorbable nylon suture with two square knots. A modified

Robert-Jones dressing was applied and the PICC was then maintained with SID bandage changes and q 6 hr patency checks and flushing with heparinized (1 international unit [IU]/mL) sodium chloride. Cobalamin, folate, and feline pancreas-specific lipase (fPL) samples (after a 12 hour fast) were then collected by the applicant to evaluate for an underlying enteropathy that may be contributing to the patient's clinical signs as well as evaluation of suspected pancreatic lipase elevation given AUS findings. A serum separator tube was filled with 4 mL of whole blood and after clot formation, the sample was centrifuged at 2500 revolutions per min for 15 min. The resulting serum was separated into a no additive tube to be shipped on ice (to maintain a temperature of 4°C) to an outside laboratory. A BG was also rechecked at this time, revealing improvement of hyperglycemia (502 mg/dL). Additional diagnostics to consider at this point in the patient's care would be repeating serum ketone analysis to evaluate the status of previously noted ketonemia or to evaluate for ketonuria in-house via a ketone reagent strip. A point-of-care fPL assay would also be beneficial in confirming pancreatic lipase elevation with consideration for time efficiency.

Therapeutic interventions focused on maintaining euhydration and prevention of electrolyte imbalances, antacid and antiemetic therapy for gastroprotection and treatment of nausea that could be contributing to hyporexia, analgesia, treatment of hyperglycemia and subsequent ketosis, and serial BG monitoring. The patient was maintained in hospital with a balanced crystalloid solution with potassium chloride 20 mEq/L at 5.06 mL/kg/hr (20 mL/hr) IV, maropitant 1 mg/kg (3.95 mg) IV SID, pantoprazole 1 mg/kg (3.95 mg) IV BID, BG monitoring q 2 hr, PICC bandage changes SID, gabapentin 1.9 mg/kg (7.5 mg) PO BID, buprenorphine 0.01 mg/kg (0.04 mg) IV QID, an adjustment to insulin therapy with transition to long-acting PZI

0.63 units/kg (2.5 units) SC BID, and BID nutrition of a balanced maintenance diet prior to PZI administration. At this time, a moderate to large appetite was noted (see **Appendix E**) and a 6 hr post PZI BG nadir of 211 mg/dL was appreciated (see **Appendix D**). Close monitoring for progression of or new clinical signs such as vomiting or diarrhea was performed. Pain assessments were also performed to ensure an appropriate comfort level and to monitor for oversedation with analgesic therapy.

Case Management

After 24 hours of hospitalization and supportive care with the applicant's service (October 6th, 2022), the patient appeared to have bright mentation with no pain elicited on abdominal palpation (see **Appendix A**). Vital parameters were within normal limits and a moderate appetite was appreciated while offering a balanced maintenance diet (see **Appendix E**). A BG nadir of 232 mg/dL was also noted (see **Appendix D**) with no significant findings on CBC, chemistry, or VBG with the exception of improvement of hyperglycemia (223 mg/dL on chemistry) (see **Appendix B**). Urinalysis returned at that time and revealed glucosuria (500 mg/dL [N=0 mg/dL]) (see **Appendix F**), which was to be expected in a patient with DM, and otherwise showed no significant findings. The patient remained in hospital for an additional 24 hours to ensure the patient's moderate appetite remained consistent. Medical therapy remained static with the exception of a decrease of the balanced crystalloid solution with potassium chloride 20 mEq/L to 3.8 mL/kg/hr (15 mL/hr) IV, as the patient maintained euhydration.

After an additional 24 hours of hospitalization (October 7th, 2022), no concerning changes were appreciated on PE (see **Appendix A**). Vital parameters remained within normal limits and the patient remained comfortable, bright, and alert with a moderate to large appetite

(see **Appendix E**). Given the patient's clinical progress and aforementioned improvement of hyperglycemia, the patient was discharged for continued at home care. Diagnostics prior to discharge included continued BG monitoring q 2 hr (see **Appendix D**). Although the patient's hyperglycemia had improved post admittance, BG pre-insulin administration at 0800 was above the desired threshold with PZI administration (>216 mg/dL) at 456 mg/dL and it was anticipated that an increase in dose would be required in the future if this remained persistent (Rand & Gottlieb, 2017). The patient was discharged with the following medical therapy to be continued until otherwise directed: gabapentin 1.9 mg/kg (7.5 mg) PO BID PRN, buprenorphine 0.015 mg/kg (0.06 mg) administered on the mucous membranes TID PRN, maropitant 2 mg/kg (8 mg) PO SID, PZI 0.63 units/kg (2.5 units) SC BID, and instructions to feed any palatable maintenance balanced diet BID prior to PZI administration. Instructions for continued medical therapy and at-home monitoring were provided to the owner (see **Appendix G**). A one month post discharge recheck examination with fructosamine level was recommended.

Final Outcome

Pending diagnostics revealed cobalamin and folate were within normal limits (see **Appendix H**) and no growth was evident on urine culture. However, fPL was significantly elevated (50 micrograms/L [µg/L], [N=0.0-3.5 µg/L]), providing further evidence of pancreatitis.

Recheck evaluations were elected with the patient's rDVM and limited medical updates were provided to the applicant's facility. The patient had reportedly experienced multiple incidences of hyporexia post hospitalization. Fructosamine upon initial post-hospitalization recheck was not consistent with appropriate control of DM (625 micromoles/L [µmol/L], [N=191-349 µmol/L]), and chronic pancreatitis was suspected. The patient's medical therapy

was not adjusted, with the exception of the PZI dose, which was increased to 0.76 units/kg (3.0 units) SC BID. Multiple additional fructosamine levels were provided, revealing consistently high values (629 µmol/L 1/23/23 and 702 µmol/L 4/4/23). PZI was further increased to 0.88 units/kg (3.5 units) SC BID on 1/23/23 and mirtazapine 0.5 mg/kg (2 mg) transdermally SID was initiated on 4/4/23 due to consistent hyporexia. Further updates on the patient's status have yet to be received.

Discussion

DM is characterized as persistent hyperglycemia due to insulin deficiency. Absolute or transient insulin deficiency is caused by an insufficiency in secretion of insulin from pancreatic beta-cells (Rand & Gottlieb, 2017). The etiology of pancreatic beta-cell failure classifies the type of DM. Current classifications in animals are based on human diabetes and are categorized into four types: type 1, type 2, gestational diabetes, and other specific types such as genetic syndromes (Kharroubi & Darwish, 2015). Type 2 diabetes is described as reduced insulin sensitivity and an inability of pancreatic beta-cells to compensate for said insulin resistance, resulting in hyperglycemia (Gottlieb & Rand, 2018). Although the mechanisms of beta-cell impairment are not fully understood, examples of risk factors of type 2 diabetes include, but are not limited to, obesity, increased age, steroid use, and physical inactivity. With type 2 diabetes accounting for the majority of cases in the feline population (Gottlieb & Rand, 2018), it is presumed that this patient suffers from type 2 diabetes based on a diagnosis of exclusion with a lack historical medical evidence to suggest otherwise (such a neoplasia, hyperadrenocorticism, and acromegaly). Clinical signs of DM include weight loss despite a normal or increased appetite, as insulin resistance causes a reduction in utilization of glucose in peripheral tissues

(Rand & Gottlieb, 2017), resulting in fats and proteins being broken down to feed glucose-starved cells. Osmotic diuresis secondary to hyperglycemia and glucosuria leads to the compensatory clinical changes of polyuria and polydipsia as well (Gottlieb & Rand, 2018). In severely unregulated DM, free fatty acid release as a response to cell starvation leads to the hepatic production of acetoacetate and beta-hydroxybutyrate (ketone bodies) as an alternative energy source, promoting ketogenesis (Rudloff, 2017). The accumulation of acidic ketone bodies can then disrupt acid-base balance, resulting in a metabolic acidosis.

Pancreatitis is described as inflammation of the pancreas that can be acute or chronic in nature with varying degrees of severity. Although >95% of feline pancreatitis cases are idiopathic, pancreatitis has been associated with concurrent diseases such as DM, cholangitis, enteropathies, and hepatic lipidosis (Forman et al., 2021). Uncommon etiologies of pancreatitis include trauma, drug-induced pancreatitis, pancreatic neoplasia, and parasitic or viral infections (Forman et al., 2021). While this patient's pancreatitis was idiopathic, pancreatitis is an established concurrent disease seen with DM and the relationship between the two is thought to be reciprocal, with each disease allowing for predisposition to the other (Xenoulis & Fracassi, 2022). Clinical signs of pancreatitis are non-specific and may include abdominal discomfort, vomiting, diarrhea, anorexia, and lethargy and management requires symptomatic treatment, rehydration, analgesia, and nutritional support. Despite prolonged symptomatic and analgesic therapy in this patient, continued diabetic dysregulation and hyporexia were reported and chronic pancreatitis was highly suspected. Acute and chronic pancreatitis cannot be differentiated based on clinical manifestation, however either has the potential to present with comorbidities and complications that increase the risk of mortality (Ruaux, 2017).

Diagnostically, CBC, chemistry, and VBG were vital in evaluating for hyperglycemia, a metabolic acidosis, anemia, leukocytosis, dehydration, and electrolyte imbalances that can be present with unregulated, acidotic, and/or ketotic diabetics. These parameters are also essential to rule out other common concurrent abnormalities such as azotemia and elevated triglycerides, cholesterol, and liver enzymes (O'Brien, 2017). Establishing ketone bodies either via urine, serum, or plasma and severity of ketosis is imperative in understanding severity of glycemic dysregulation, the presence of which was not unexpected in this patient given their past occurrences of diabetic ketoacidosis and presenting clinical sign of hyporexia. Urinalysis and culture were utilized to exclude the presence of a urinary tract infection and cobalamin and folate were utilized to evaluate for an underlying enteropathy, as either can contribute to glycemic dysregulation in diabetics. Imaging, including thoracic radiographs and AUS to assist in excluding neoplasia (given the patient's increased age), was a principal indicator of prognosis in this patient. Ultrasonographically, the hypoechoic and irregularly margined pancreas visualized was anticipated given the noted abdominal discomfort, although abdominal pain is not commonly documented in cats with pancreatitis (Ruaux, 2017). In addition to AUS, elevated fPL was also a diagnostic indicator of pancreatitis, as this assay measures lipase derived from the acinar cells of the pancreas (Schnauß et al., 2019).

Treatment in this patient focused on insulin therapy to improve hyperglycemia and decrease ketone body concentrations. As the patient was not acidotic, insulin therapy involved short-acting regular insulin IM, followed by long-acting insulin (PZI) SC for long-term therapy once the patient was euhydrated. Serial monitoring of BG via a PICC was imperative to making insulin therapy adjustments PRN, with consideration for the addition of dextrose

supplementation should a hypoglycemic event have occurred. A balanced crystalloid solution with potassium chloride IV was initiated to correct dehydration and avoid potential hypokalemia, as insulin can drive potassium intracellularly, lowering the serum concentration of the electrolyte (O'Brien, 2017). Gastric acid suppression and antiemetic therapy, with pantoprazole and maropitant, was implemented due to the presenting hyporexia, likely secondary to pancreatitis. Multi-modal analgesia with gabapentin and buprenorphine was also necessary, as this patient also exhibited signs of mild to moderate pain secondary to pancreatitis. An additional therapeutic modality for consideration in this patient would include a diet that is appropriate for diabetic maintenance and pancreatitis such as a lower fat, high protein, and low carbohydrate diet, however this may not be pragmatic in a patient exhibiting hyporexia without the ability of enteral feeding. In consideration for long-term care, monitoring of the serum glycated protein fructosamine was elected to avoid potential stress induced hyperglycemia that may cause misinterpretation of diabetic control (Gal et al., 2017), which unfortunately has shown continued glycemic dysregulation due to suspected chronic pancreatitis.

Understanding of the inflammatory nature of this patient's disease process and identifying analgesic need allowed for proposal of a multi-modal analgesic plan by the applicant. Pain management is an essential aspect to increasing the probability of a positive outcome by relieving clinical signs while also providing empathetic care. In addition, the applicant's ability to perform advanced technical skills, such as placement of a PICC, allowed for low stress handling and the necessary serial BG monitoring of a patient suffering from diabetic ketosis without the need for repeated venipuncture.

References

Forman, M. A., Steiner, J. M., Armstrong, P. J., Camus, M. S., Gaschen, L., Hill, S. L., Mansfield, C. S., & Steiger, K. (2021). ACVIM consensus statement on pancreatitis in cats. *Journal of veterinary internal medicine*, *35*(2), 703–723. https://doi.org/10.1111/jvim.16053

Gal, A., Trusiano, B., French, A. F., Lopez-Villalobos, N., & MacNeill, A. L. (2017). Serum Fructosamine Concentration in Uncontrolled Hyperthyroid Diabetic Cats Is within the Population Reference Interval. *Veterinary sciences*, *4*(1), 17. https://doi.org/10.3390/vetsci4010017

Gottlieb, S., & Rand, J. (2018). Managing feline diabetes: current perspectives. *Veterinary medicine (Auckland, N.Z.)*, *9*, 33–42. https://doi.org/10.2147/VMRR.S125619

Kharroubi, A. T., & Darwish, H. M. (2015). Diabetes mellitus: The epidemic of the century. *World journal of diabetes*, *6*(6), 850–867. https://doi.org/10.4239/wjd.v6.i6.850

O'Brien M. (2017). Diabetic ketoacidosis and hyperglycemic hyperosmolar syndrome. In S. J. Ettinger, E. C. Feldman, & E. Cote (Eds.), *Textbook of Veterinary Internal Medicine* (Vols. 1-2) (pp. 3380-3433). Saunders.

Rand J. & Gottlieb S. A. (2017). Feline diabetes mellitus. In S. J. Ettinger, E. C. Feldman, & E. Cote (Eds.), *Textbook of Veterinary Internal Medicine* (Vols. 1-2) (pp. 10271-10366). Saunders.

Ruaux C. G. (2017). Feline pancreatitis. In S. J. Ettinger, E. C. Feldman, & E. Cote (Eds.), *Textbook of Veterinary Internal Medicine* (Vols. 1-2) (pp. 9818-9841). Saunders.

Rudloff E. (2017). Diabetic ketoacidosis in the cat: Recognition and essential treatment. *Journal*

of feline medicine and surgery, *19*(11), 1167–1174. https://doi.org/10.1177/1098612X17735762

Schnauß, F., Hanisch, F., & Burgener, I. A. (2019). Diagnosis of feline pancreatitis with SNAP fPL and Spec fPL. *Journal of feline medicine and surgery*, *21*(8), 700–707. https://doi.org/10.1177/1098612X18796624

Xenoulis, P. G., & Fracassi, F. (2022). Feline Comorbidities: Clinical perspective on diabetes mellitus and pancreatitis. *Journal of feline medicine and surgery*, *24*(7), 651–661. https://doi.org/10.1177/1098612X221106355

Appendix A

Physical Examination Findings

10/04/22

Physical examination:
Quiet, alert and responsive. Hydration - estimated 5-8% dehydrated.
Eyes, Ears, Nose and Throat: No discharge or odor observed AU. No discharge or sneezing observed.
Peripheral Lymph Nodes: No abnormalities in size or contour observed on palpation of peripheral lymph nodes.
Heart and Lungs: No murmur or arrhythmias observed; strong, synchronous femoral pulses. Mild tachypnea; No adventitial sounds on auscultation. no crackles or wheezes; clear lung fields bilaterally.
Abdomen: No discomfort or pain observed on palpation.
Urogenital: No significant findings.
Musculoskeletal: Body condition score (BCS) 3/9. Ambulatory on all 4 limbs. Gait not assessed
Integument: Hair coat and skin in good condition. No obvious ectoparasites.
Rectal: Not performed.

10/05/22

Physical examination:
Quiet, alert and responsive. Well hydrated with moist, pink mucous membranes, and normal capillary refill time.
Eyes, Ears, Nose and Throat: Clear eyes with no ocular discharge. Fundic exam normal. No aural discharge. Both nares patent and clear of discharge. No significant abnormalities on oral exam, aside from periodontal disease.
Peripheral Lymph Nodes: Normal.
Heart and Lungs: No murmurs or arrhythmias. Strong and synchronous pulses. Normal respiratory rate and effort with clear lung sounds bilaterally.
Abdomen: Mild to moderate pain elicited on palpation. No obvious masses.
Urogenital: Kidneys smooth and symmetrical. Moderately sized, soft bladder.
Musculoskeletal: Ambulatory on all 4 limbs. Generalized muscle wasting. BCS 4/9.
Integument: Normal coat. Normal skin.
Rectal: Not performed.

10/06/22

Physical examination:
Bright, alert and responsive. Well hydrated with moist, pink mucous membranes, and normal capillary refill time.

Eyes, Ears, Nose and Throat: Clear eyes with no ocular discharge. Fundic exam normal. No aural discharge. Both nares patent and clear of discharge. No significant abnormalities on oral exam, aside from periodontal disease.
Peripheral Lymph Nodes: Normal.
Heart and Lungs: No murmurs or arrhythmias. Strong and synchronous pulses. Normal respiratory rate and effort with clear lung sounds bilaterally.
Abdomen: Soft and non-painful. No obvious masses.
Urogenital: Kidneys smooth and symmetrical. Moderately sized, soft bladder.
Musculoskeletal: Ambulatory x 4. Generalized muscle wasting. BCS 4/9.
Integument: Normal coat. Normal skin.
Rectal: Not performed.

10/07/22

Physical examination:
Bright, alert and responsive. Well hydrated with moist, pink mucous membranes, and normal capillary refill time.
Eyes, Ears, Nose and Throat: Clear eyes with no ocular discharge. Fundic exam normal. No aural discharge. Both nares patent and clear of discharge. No significant abnormalities on oral exam, aside from periodontal disease.
Peripheral Lymph Nodes: Normal.
Heart and Lungs: No murmurs or arrhythmias. Strong and synchronous pulses. Normal respiratory rate and effort with clear lung sounds bilaterally.
Abdomen: Soft and non-painful. No obvious masses.
Urogenital: Kidneys smooth and symmetrical. Moderately sized, soft bladder.
Musculoskeletal: Ambulatory x 4. Generalized muscle wasting. BCS 4/9.
Integument: Normal coat. Normal skin.
Rectal: Externally normal.

Appendix B

Laboratory Results

10/04/22

Venous Blood Gas (VBG)			
Test	**Results**	**Unit**	**Reference Range**
pH	7.378		7.230-7.423
partial pressure of carbon dioxide (PCO2)	41.8	millimeters of mercury (mmHg)	42.1 ± 4.4
partial pressure of oxygen (PO2)	57.6	millimeters of mercury (mmHg)	55 ± 9.6
bicarbonate (HCO3)	24.1	mmol/L	18.1-26.3
base excess (BE)	-1.1	mmol/L	-5.7 ± 5
Hct	32	%	31-48
sodium (Na)	151.2	mmol/L	149.8-155.7
potassium (K)	4.09	mmol/L	3.19-4.76
ionized calcium (Ca++)	1.26	mmol/L	1.19-1.35
chloride (Cl)	114	mmol/L	113-121
glucose	614	mg/dL	75-120
lactate	0.8	mmol/L	0.5-5.23
anion gap	17.2	mmol/L	15.4-23.4

CBC			
Test	**Results**	**Unit**	**Reference Range**
RBC	6.72	million/microliter (M/µL)	6.54-12.2
Hct	35	%	30.3-52.3
Hgb	9.9	g/dL	9.8-16.2
MCV	41	femtoliters (fL)	35.9-53.1
MCH	14.2	picograms (pg)	11.8-17.3
MCHC	34.7	g/dL	28.1-35.8

red blood cell distribution width (RDW)	22.6	%	15-27
reticulocytes	0.6	%	
absolute reticulocytes	34.8	kilo/microliter (K/µL)	3.0-50.0
reticulocyte-Hgb	16	picograms (pg)	13.2-20.8
WBC	8.82	kilo/microliter (K/µL)	2.87-17.02
% neutrophils	69	%	
% lymphocytes	13.5	%	
% monocytes	3.1	%	
% eosinophils	13.4	%	
% basophils	1	%	
neutrophils	6.09	kilo/microliter (K/µL)	2.30-10.29
lymphocytes	1.19	kilo/microliter (K/µL)	0.92-6.88
monocytes	0.27	kilo/microliter (K/µL)	0.05-0.67
eosinophils	1.18	kilo/microliter (K/µL)	0.17-1.57
basophils	0.09	kilo/microliter (K/µL)	0.01-0.26
platelets	342	kilo/microliter (K/µL)	151-600
mean platelet volume (MPV)	17	femtoliters (fL)	11.4-21.6
plateletcrit (PCT)	0.58	%	0.17-0.86

Chemistry			
Test	**Results**	**Unit**	**Reference Range**
glucose	634	mg/dL	71-159
CREA	1.5	mg/dL	0.8-2.4
BUN	27	mg/dL	16-36
BUN/CREA	17		
phosphorus	5.5	mg/dL	3.1-7.5
calcium	11	mg/dL	7.8-11.3
total protein	8.6	g/dL	5.7-8.9
albumin	3.3	g/dL	2.3-3.9
globulin	4.9	g/dL	2.8-5.1
albumin/globulin	0.6		
ALT	42	units/L (U/L)	12-130

ALKP	75	units/L (U/L)	14-111
GGT	0	units/L (U/L)	0-4
T.Bili	0.8	mg/dL	0.0-0.9
cholesterol	179	mg/dL	65-225
amylase	679	units/L (U/L)	500-1500
lipase	1,106	units/L (U/L)	100-1400
sodium	159	mmol/L	150-165
potassium	4.4	mmol/L	3.5-5.8
sodium/potassium	36		
chloride	113	mmol/L	112-129
osmolality	353	mmol/kg	

Ketone Analysis			
Test	**Results**	**Unit**	**Reference Range**
serum ketone analysis	15	mg/dL	0

10/06/22

Venous Blood Gas (VBG)			
Test	**Results**	**Unit**	**Reference Range**
pH	7.377		7.230-7.423
partial pressure of carbon dioxide (PCO2)	46.5	millimeters of mercury (mmHg)	42.1 ± 4.4
partial pressure of oxygen (PO2)	45.8	millimeters of mercury (mmHg)	55 ± 9.6
bicarbonate (HCO3)	26.2	mmol/L	18.1-26.3
base excess (BE)	1	mmol/L	-5.7 ± 5
Hct	33.4	%	31-48
sodium (Na)	153.7	mmol/L	149.8-155.7
potassium (K)	3.37	mmol/L	3.19-4.76
ionized calcium (Ca++)	1.24	mmol/L	1.19-1.35
chloride (Cl)	114	mmol/L	113-121

glucose	197	mg/dL	75-120
lactate	0.66	mmol/L	0.5-5.23
anion gap	16.9	mmol/L	15.4-23.4

CBC			
Test	**Results**	**Unit**	**Reference Range**
RBC	6.64	million/microliter (M/µL)	6.54-12.2
Hct	34.1	%	30.3-52.3
Hgb	10.2	g/dL	9.8-16.2
MCV	41.8	femtoliters (fL)	35.9-53.1
MCH	14.7	picograms (pg)	11.8-17.3
MCHC	35.1	g/dL	28.1-35.8
red blood cell distribution width (RDW)	19.2	%	15-27
reticulocytes	0.3	%	
absolute reticulocytes	14.4	kilo/microliter (K/µL)	3.0-50.0
reticulocyte-Hgb	15.8	picograms (pg)	13.2-20.8
WBC	9.79	kilo/microliter (K/µL)	2.87-17.02
% neutrophils	67	%	
% lymphocytes	20.9	%	
% monocytes	5.1	%	
% eosinophils	6.2	%	
% basophils	0.8	%	
neutrophils	6.55	kilo/microliter (K/µL)	2.30-10.29
lymphocytes	2.05	kilo/microliter (K/µL)	0.92-6.88
monocytes	0.5	kilo/microliter (K/µL)	0.05-0.67
eosinophils	0.61	kilo/microliter (K/µL)	0.17-1.57
basophils	0.08	kilo/microliter (K/µL)	0.01-0.26
platelets	163	kilo/microliter (K/µL)	151-600
mean platelet volume (MPV)	18.1	femtoliters (fL)	11.4-21.6
plateletcrit (PCT)	0.19	%	0.17-0.86

Chemistry			
Test	**Results**	**Unit**	**Reference Range**
glucose	223	mg/dL	71-159
CREA	1.7	mg/dL	0.8-2.4
BUN	20	mg/dL	16-36
BUN/CREA	12		
phosphorus	5.2	mg/dL	3.1-7.5
calcium	10.6	mg/dL	7.8-11.3
total protein	8.4	g/dL	5.7-8.9
albumin	3.1	g/dL	2.3-3.9
globulin	5.1	g/dL	2.8-5.1
albumin/globulin	0.6		
ALT	73	units/L (U/L)	12-130
ALKP	41	units/L (U/L)	14-111
GGT	1	units/L (U/L)	0-4
T.Bili	0.5	mg/dL	0.0-0.9
cholesterol	219	mg/dL	65-225
amylase	744	units/L (U/L)	500-1500
lipase	852	units/L (U/L)	100-1400
sodium	158	mmol/L	150-165
potassium	4.6	mmol/L	3.5-5.8
sodium/potassium	34		
chloride	116	mmol/L	112-129
osmolality	316	mmol/kg	

Appendix C

Diagnostic Imaging Results

10/04/22

Thoracic radiograph report:
The pulmonary parenchyma is unremarkable. The cardiac silhouette is rounded on the ventrodorsal projection, possibly secondary to pericardial fat accumulation. The pulmonary vasculature and pleural space are unremarkable. There is no evidence of thoracic lymphadenopathy. The trachea and region of the esophagus are unremarkable.

10/5/22

Abdominal ultrasound report:
Biliary system: Normal appearance of gallbladder wall and bile. The rest of the biliary tree is not visible.
Liver: Normal appearance. The vasculature is normal.
Spleen: Normal appearance of parenchyma and vasculature.
Left kidney: No pelvic dilation. There is mild, amorphous central/pelvic mineralization. The left kidney is 3.68 cm in length.
Right kidney: No pelvic dilation or mineralization. The right kidney is 4.39 cm in length.
Urinary bladder: Normal appearance of urine and bladder wall.
Left adrenal gland: Normal size and appearance. Width: 0.38 cm.
Right adrenal gland: Normal size and appearance. Width: 0.30 cm.
Stomach: Normal wall layering. The stomach is empty.
Small intestine: Normal small intestinal wall layering. The duodenal wall measures 0.29 cm. An additional small intestinal (jejunal) loop wall measures 0.25 cm. The ileal wall measures 0.31 cm. The mesentery appears normal.
Colon: Normal wall layering. The colonic wall measures 0.14 cm in thickness. There are feces and gas in the lumen.
Pancreas: The pancreas is diffusely enlarged and hypoechoic with an irregular contour.
Lymph nodes: Normal appearance.
Additional findings: There is scant, anechoic peritoneal fluid.

Appendix D

Blood Glucose Monitoring and Interventions

10/04/22

Time	Blood Glucose Concentration	Intervention
1900	634 mg/dL	
2300	703 mg/dL	regular insulin human injection 0.126 units/kg (0.5 units) IM

10/05/22

Time	Blood Glucose Concentration	Intervention
0100	251 mg/dL	
0300	222 mg/dL	
0500	440 mg/dL	regular insulin human injection 0.126 units/kg (0.5 units) IM
0700	468 mg/dL	
1000	502 mg/dL	protamine zinc recombinant human insulin 0.63 units/kg (2.5 units) SC
1200	595 mg/dL	
1400	573 mg/dL	
1600	211 mg/dL	
1800	378 mg/dL	
2000	451 mg/dL	
2200	488 mg/dL	protamine zinc recombinant human insulin 0.63 units/kg (2.5 units) SC
2400	518 mg/dL	

10/06/22

Time	Blood Glucose Concentration	Intervention
0200	413 mg/dL	
0400	526 mg/dL	
0600	457 mg/dL	

0800	486 mg/dL	protamine zinc recombinant human insulin 0.63 units/kg (2.5 units) SC
1000	422 mg/dL	
1200	238 mg/dL	
1400	232 mg/dL	
1600	351 mg/dL	
1800	454 mg/dL	
2000	506 mg/dL	protamine zinc recombinant human insulin 0.63 units/kg (2.5 units) SC
2200	451 mg/dL	
2400	391 mg/dL	

10/7/22

Time	**Blood Glucose Concentration**	**Intervention**
0200	299 mg/dL	
0400	351 mg/dL	
0600	490 mg/dL	
0800	456 mg/dL	protamine zinc recombinant human insulin 0.63 units/kg (2.5 units) SC

Appendix E

Clinical and Vital Parameters

10/05/22

Clinical Parameters		
		Time
attitude	quiet, alert, and responsive	
appetite	moderate to large, balanced maintenance diet	1100, 2200
vomiting	none	
diarrhea	none	
urination	normal	
pain	mild	

10/06/22

Vital Parameters		
		Unit
Doppler blood pressure	140	millimeters of mercury (mmHg)
temperature	99.9	degrees Fahrenheit (℉)
heart rate	180	bpm
pulse rate	180	pulse rate per minute
respiratory rate	32	respirations per minute
respiratory effort	none	
capillary refill time (CRT)	1-2	seconds
mucous membrane color	pink	
weight	4.12	kg

Clinical Parameters		
		Time
attitude	quiet, alert, and responsive	

appetite	moderate, balanced maintenance diet	0800, 2000
vomiting	none	
diarrhea	none	
urination	normal	
pain	none	

10/07/22

Vital Parameters		
		Unit
Doppler blood pressure	128	millimeters of mercury (mmHg)
temperature	100.1	degrees Fahrenheit (℉)
heart rate	200	bpm
pulse rate	200	pulse rate per minute
respiratory rate	36	respirations per minute
respiratory effort	none	
capillary refill time (CRT)	1-2	seconds
mucous membrane color	pink	
weight	4	kg

Clinical Parameters		
		Time
attitude	bright, alert, and responsive	
appetite	Moderate to large, balanced maintenance diet	0800
vomiting	none	
diarrhea	none	
urination	normal	
pain	none	

Appendix F

Urinalysis Results

Urinalysis	
Test	**Results**
color	yellow
clarity	clear
specific gravity	1.023
pH	7.5
urine protein	negative
glucose	2+ (500 mg/dL)
ketones	trace
blood/hemoglobin	negative
bilirubin	negative
urobilinogen	normal
white blood cells	0-2/HPF
red blood cells	0-2/HPF
bacteria	none
epithelial cells	rare (0-1/HPF)
mucus	none
casts	none
crystals	none

Appendix G

Discharge Instructions

10/7/22

Barnum was initially evaluated on 10/4/22 for a poor appetite. On presentation, he was dehydrated with profound hyperglycemia. He was found to be in the early stages of diabetic ketosis, with evidence of pancreatitis on abdominal ultrasound. Pancreatitis is inflammation of the pancreas and can cause gastrointestinal upset, dehydration, lethargy, and poor appetite. It can also make diabetic control difficult, as is the case with Barnum. He was treated with intravenous fluids, insulin, and gastroprotectant medications, which improved his hyperglycemia and poor appetite. His hyperglycemia and appetite remained improved this morning, so he is ready to be discharged.

MEDICATIONS:

Continue protamine zinc insulin as previously directed. No change in dosing or dosing schedule is needed. Remember to never increase the dose of insulin without contacting a veterinarian first. Reducing the dose, however, is advised if Barnum refuses food at the time of feeding and has not eaten within the past 12 hours. In this case, do not give any insulin and seek veterinary advice.

Barnum's blood sugar prior to discharge was moderately elevated, so please be sure he gets his insulin when he returns home this evening. If he shows increased water intake or increased volume/frequency of urine produced, please contact us, as we may need to increase his insulin dose.

-protamine zinc insulin (40U/ml): Continue to give 2.5 units underneath the skin twice daily after a meal.

-maropitant (16 mg tablets): Give 1/2 of a tablet (8 mg) by mouth once daily as needed (if Barnum is vomiting or his appetite is poor). It can be given with or without food.

-buprenorphine (0.3 mg/ml): Give 0.2 ml (0.06 mg) by injecting the syringe into the cheek pouch 3 times daily, as needed (if Barnum appears uncomfortable, as pancreatitis can cause abdominal pain). It does not need to be given with food; it is absorbed across the gums. This medication reduces pain. Side effects of this medication may include excessive sedation or constipation.

-gabapentin (50 mg/ml): Give 0.15 ml (7.5 mg) by mouth twice daily as needed if Barnum

appears uncomfortable.

DIET:
No change in Barnum's diet is warranted at this time. Please be sure his appetite is at the level it was prior to his illness.

FOLLOW-UP:
A recheck examination is recommended in 1 month for a fructosamine level.

ADDITIONAL INSTRUCTIONS:

Barnum should be monitored for vomiting, diarrhea, a poor appetite, lethargy, or signs of pain such as aggression, vocalization, and abnormal posture. If you are at all concerned with his condition, please do not hesitate to contact us.

Appendix H

Feline Gastrointestinal Laboratory Parameters

Test	Results	Unit	Reference Range
cobalamin	853	nanogram/dL (ng/dL)	276-1425
folate	18.7	microgram/dL (µg/dL)	8.9-19.9