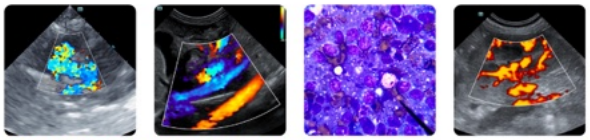

PATIENT

Zeus Spada

SPECIES

Canine

BREED

Pug

SEX

Neutered male

AGE

8 ½ years

WEIGHT

23 lbs

INTERPRETED BY

Remo Lobetti, BVSc, MMedVet (Med), PhD, Dipl. ECVIM

IMAGING PERFORMED BY

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PRESENTING CLINICAL SIGNS

History: Recently diagnosed diabetic. At the time of diagnosis, also diagnosed with pancreatitis. Recent dx of MCT on left shoulder (SQ). Has chronic ocular changes (KCS, pigmentary changes) On Cyclosporine. Started treatment with insulin (NPH); recently increased to 7U BID (0.7U/kg). Continues to be unregulated PU/PD with weight loss. Appetite great, signs of pancreatitis resolved. Performed AUS and LDDS to assess liver enzymes/adrenals and screen.
Abnormal PE/Chem/CBC/UA Results: PE: weight loss, chronic changes to eyes, otherwise NSF. Pre-diagnosis 2/7/2025 ALP 380 1 week post diagnosis @ ER Clinic 10/7/25: CBC- WNL, Chem- BG 541, Alb 2.3, Glob 4.5, ALT 284, ALP 3912, GGT 37, Tbili 0.6, Chol 355, spc Cpl 360 (H but improved) 11/5/25- Fructosamine 418, CBC- WNL, Chem BG 439, Alb 2.6, ALT 185, ALP 872, 12/3/25- Fructosamine 540, ALT 208, ALP 1407

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder is full with a normal thickness and smooth appearance of the wall. Normal anechoic urine with no sediment or uroliths evident.

Normal appearance of the trigone area, proximal urethra, and iliac blood vessels.

Normal appearance and size of the iliac lymph nodes. Ureters not visualized, which can be considered a normal finding.

Normal renal size (left measured 4.9 cm, right measured 4.9 cm), increased echogenic appearance of the cortex, but maintained a normal cortico-medullary differentiation, pelvis, and capsule. No infarcts, mineralization or renoliths evident.

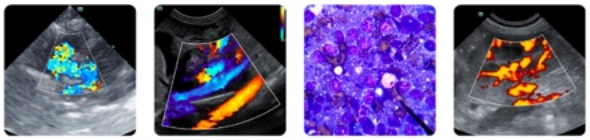
Small, hypoechogenic prostate.

Adrenal Glands

The left adrenal gland is normal in shape, echogenic appearance, size, position, and appearance of the visible peri-adrenal vasculature. Left adrenal gland measured 0.49 cm in width. The right adrenal gland was not clarly visualized, but appears to be of normal shape, echogenic appearance and size.

Spleen

Normal size and echogenic appearance. Smooth homogenous parenchyma and regular curvilinear capsule. Normal volume of the splenic vasculature without any overt congestion or thrombosis evident. No inflammatory, neoplastic, infarction, or infiltrative changes evident. The spleen measured 0.9 cm in width.



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Liver

The liver was enlarged with rounded edges, diffuse increased echogenic appearance, normal portal markings, and regular curvilinear capsule. No nodules or masses evident. Normal appearance of the hepatic and portal vasculature.

Gallbladder

The gallbladder is full containing normal anechoic bile. Normal thickness and echogenic appearance of the wall. Normal size and appearance of the cystic and common bile duct.

Gastrointestinal

Normal appearance of the stomach, duodenum, small intestine, ileo-cecal junction, and colon with no loss of layering, 1:3 muscularis to mucosa ratio, normal wall thickness and peristaltic activity, and no distension of the lumen. A moderate amount of ingesta is present in the stomach compatible with a recent meal.

Pancreas

The visible sections of the pancreas are of normal size and echogenic appearance with a regular capsule. Normal echogenic appearance of the mesentery and fat surrounding the pancreas.

Free Abdomen

Normal mesenteric lymph nodes.

No ascites evident.

ULTRASONOGRAPHIC FINDINGS

- Hepatopathy.
- Age related renal changes versus early chronic kidney disease.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The most likely etiology for the hepatopathy would be metabolic secondary to the diabetes.

On this ultrasound there is no obvious etiology for the poorly controlled diabetes.

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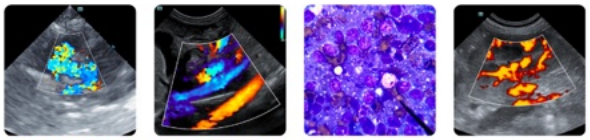

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TROUBLESHOOTING PERSISTENT HYPERGLYCAEMIA IN TREATED DIABETICS

Persistent hyperglycaemia is a frustrating and all too common problem in diabetic dogs and cats treated with insulin with numerous factors contributing to poor glucose regulation. Some of these factors are related to management, such as improper insulin administration, while others have a physiological basis, such as biologic variability of insulin action.

Identifying persistent hyperglycaemia

Diabetic dogs and cats with persistent hyperglycaemia are typically classified as poorly regulated diabetics. Owners may report that clinical signs of diabetes persist and that the recommended at-home treatment is not working as well as expected. Clinical examination may reveal findings compatible with diabetes, such as hepatomegaly, cataracts, and poor body condition. Poor glycaemic control is reflected in the laboratory evaluation as elevated fructosamine levels and abnormal glucose curves.

Once a diabetic patient is identified as having persistent hyperglycaemia, careful review of the history, clinical examination, and current therapeutic protocol may reveal possible causes or suggest a possible diagnostic work-up. A sequential approach to evaluating persistent hyperglycaemia patients is warranted bearing in mind that problems related to diabetes management are the most frequent causes of persistent hyperglycaemia. For this reason, it is important to completely rule out problems with the management protocol before beginning an extensive investigation to search for additional medical problems.

Patient history

It is important to determine whether some factor(s) related to the pet's home environment or daily habits might be responsible for the poor diabetes control. In people, glycaemic control can be upset by stress or emotional strain as well as seemingly innocuous departures from the patient's ordinary routine, such as unusual exposure to sunlight or increased physical activity. Presumably, similar factors could affect diabetic control in dogs and cats; so careful questioning of the pet's owner is warranted. Hunting or service dogs, for example, might experience increased insulin needs when working. Similarly, it is conceivable that a cat with a preference for a particular windowsill might absorb insulin more rapidly on sunny days. In addition to routine questions about appetite, water consumption, urinary and defaecation habits, and general attitude, the owner should be questioned about the diet, administration of non-prescribed medications or supplements, changes in the home environment or any unusual events. Owners often do not realize the importance of these factors and will not offer the information unless specifically asked.

Non-prescribed dietary changes, feeding excessive amounts of treats or table scraps, and some dietary supplements all have the potential to alter daily caloric intake. Some herbal medications have been associated with glycaemic dysregulation in people and might have the same effects in animals. Self-prescribed medications or medications provided by another veterinarian, especially topical preparations, may not be seen as problematic by owners. Topical ocular or otic medications containing steroids may produce systemic effects in some dogs and cats and antagonize diabetes regulation. Changes in the home environment or daily routine might also disrupt glucose control. Some events are clearly stressors on the patient, such as new pets or people in the household, a recent move to a new home, recent illness, or surgery. While it is important to consider any change in the patient's environment as potentially contributing to persistent hyperglycaemia, it is equally important to resist placing too much emphasis on relatively minor changes unless all other causes of persistent hyperglycaemia have been thoroughly investigated and ruled out.

Treatment protocol

This entails evaluation of all aspects of therapy including the insulin protocol, diet therapy, and management of any concurrent illness. When evaluating the insulin protocol, it is important to establish

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that the animal is receiving insulin at the prescribed dose and that the injection is performed properly. The first step is to confirm that the insulin and syringes being used are the prescribed products. Mismatch between the insulin product and the syringe used to administer it (e.g. using 100 IU syringes to administer 40 IU insulin) is a relatively common error that can lead to profound problems with diabetes regulation. Use of non-prescribed, degraded or out-dated insulin can be potential causes for poor glycaemic control and easily remedied. Once insulin type and syringes are ruled out as possible sources of errors, the owner's insulin handling and injection techniques should be evaluated. This can be accomplished by observing the owner draw up and inject the insulin dose. This can eliminate gross errors in insulin dosage, improper injection site selection (e.g., into adipose tissue), poor patient restraint, and related errors.

Glucose curve

If at this point, a cause for the persistent hyperglycaemia has not been found, a metabolic basis for persistent hyperglycaemia should be considered. Investigation of metabolic causes of persistent hyperglycaemia begins with glucose curve, which will document the magnitude and duration of hyperglycaemia over the course of the day. The traditional serial glucose curve is useful for monitoring blood glucose concentrations over time but recent studies have highlighted the low reproducibility of this technique, even when performed under ideal conditions. As the technique can yield vastly different results even when performed only 12-hrs apart under the exact same conditions, care must be taken not to place too much weight on a single glucose curve result. Glucose curves are usually performed in the hospital. However, techniques for teaching owners to generate at-home glucose curves in dogs and cats have also been described. Blood samples should be obtained every 1-2 hours so that rapid changes in glucose will not go undetected. In most circumstances a portable glucometer is adequate, as several models have been shown to be accurate in dogs and cats.

Insulin absorption

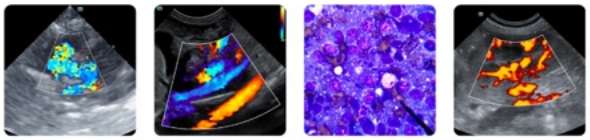
Delayed insulin absorption may lead to persistent hyperglycaemia. Insulin absorption from a subcutaneous site in humans can vary as much as 50% from day-to-day, even if the same insulin, dose, and injection site are used. As alluded to earlier, insulin absorption and action in humans can also be affected by variations in countless factors associated with daily life, including physical exercise, body temperature, and emotional state. While there are few studies in dogs and cats examining variables that alter insulin absorption and action, it is likely that there is a high degree of true day-to-day biologic variability as in humans. Biologic variability is distinct from the variability introduced by human error and the technology used for insulin administration and glucose measurement. Other potential causes of delayed insulin absorption include interference by insulin antibodies, which develop in response to exogenous insulin, and poor blood flow at the site of injection, which can occur when the injection is administered in adipose tissue. Anti-insulin antibodies have been detected in diabetic dogs and cats but there is not convincing evidence that these antibodies interfere with insulin absorption.

Somogyi reaction

This is characterized as post-hypoglycaemic hyperglycaemia and is caused by excessive insulin dosage. The patient typically has an early hypoglycaemic reaction followed by hyperglycaemia the following morning. Factors playing a role in this are the insulin dose and the patient's counter-regulatory hormones. The solution entails insulin dosage reduction by 25-50% and administering insulin twice a day.

Insulin resistance

Insulin resistance is probably the most common metabolic cause of persistent hyperglycaemia in dogs and cats but is not a specific diagnosis. By its simplest definition, insulin resistance is an inadequate biologic response to either endogenous or exogenous insulin. Insulin resistance is usually a clinical diagnosis made on the basis of persistent hyperglycaemia, an unusually high insulin requirement, and the presence of a condition associated with insulin resistance. Insulin resistance should be considered if persistent hyperglycaemia exists despite an insulin dose >2.2 IU/kg is needed to



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control hyperglycaemia. Common causes of insulin resistance include obesity, bacterial/fungal infection, and concurrent endocrinopathies. Additionally, ketosis, hyperlipidaemia, cardiac insufficiency, chronic pancreatitis, renal disease, hepatic insufficiency, and neoplasia can potentially lead to insulin resistance and poor glycaemic control. Any condition that activates counter-regulatory hormones (cortisol, catecholamines, and glucagon), such as stress, trauma, major surgery, oestrus, and pregnancy, can induce an insulin resistant state. Insulin resistance is best treated by specific treatment of the underlying disorders.

Obesity

Obesity is probably the most common cause of mild to severe insulin resistance in dogs and cats and has been shown to be associated with glucose intolerance. Obesity, hypertension, and insulin resistance commonly occur together in people with type 2 diabetes mellitus, but this metabolic complex is not well defined in veterinary patients. Insulin resistance due to obesity is reversible with sufficient weight loss and exercise.

Microbial infection

Secondary bacterial and fungal infections may cause persistent hyperglycaemia in diabetic dogs and cats. Infection may occur in any tissue but infections of the urinary tract, skin, intervertebral disc, cardiac valves, lungs, abdominal organs (especially the liver), and reproductive tract are most common in diabetic animals. Whenever possible, the organism should be identified by bacterial or fungal culture. Treatment with broad-spectrum antibiotics or antifungals should be initiated when an infection is suspected and treatment adjusted based on culture and sensitivity. Surgery may be required when an abscess has formed.

Hyperadrenocorticism

Hyperadrenocorticism is a common cause of persistent hyperglycaemia in dogs but is rare in cats. Glucocorticoids can exacerbate hyperglycaemia and impair glucose control in diabetic dogs and cats via the induction of insulin resistance in target tissues. Dogs and cats with hyperadrenocorticism often require very large amounts of insulin to maintain reasonable control of glycaemia. The diagnosis is made using adrenal testing (ACTH stimulation test, low-dose-dexamethasone suppression test) with treatment options as for non-complicated hyperadrenocorticism.

Acromegaly

Growth hormone excess can exacerbate diabetes and cause persistent hyperglycaemia in dogs and cats. Acromegaly causes severe insulin resistance, diabetes and persistent hyperglycaemia in cats. In dogs, acromegaly typically affects intact females and is caused by stimulation of growth hormone secretion from mammary tissue by exogenous or exogenous progesterone. Treatment of persistent hyperglycaemia due to growth hormone excess is difficult to accomplish in cats with pituitary tumours, which may not respond well to any therapeutic modality, but can be addressed in dogs by ovariohysterectomy or by discontinuation of exogenous progesterone treatment.

Thyroid disorders

Hyperthyroidism in cats and hypothyroidism in dogs have both been reported to be associated with insulin resistance. Cats with hyperthyroidism have fasting hyperinsulinaemia and impaired glucose tolerance, suggestive of insulin resistance. The pathophysiology underlying
insulin resistance in hypothyroidism may result from the obesity that develops in affected dogs but elevated growth hormone secretion is also known to occur in hypothyroid dogs. Treatment involves normalizing thyroid function and options are the same for non-complicated hyperthyroidism and hypothyroidism in non-diabetic cats and dogs, respectively.

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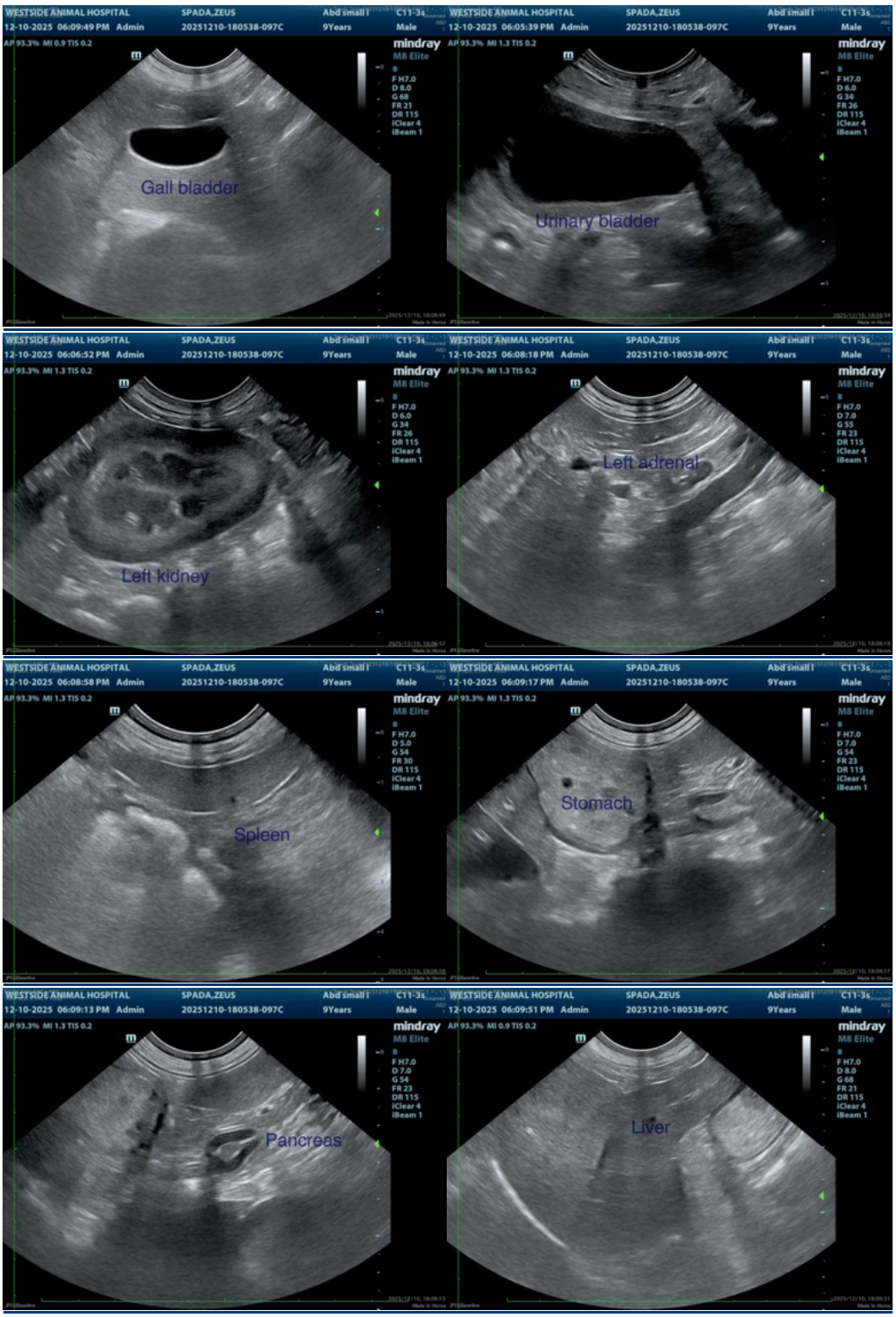

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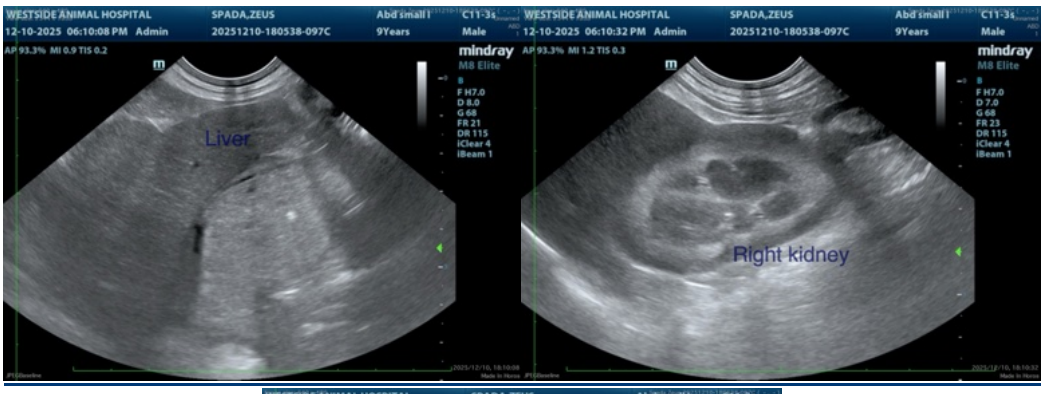

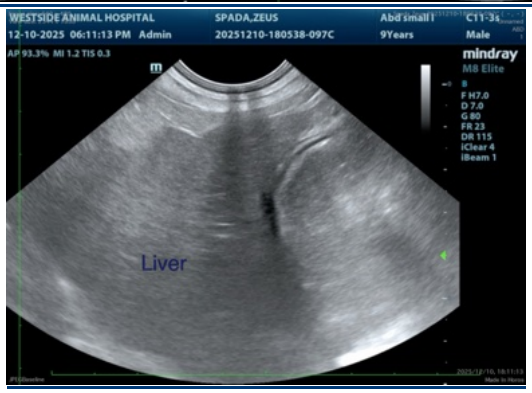

The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance please contact me.

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