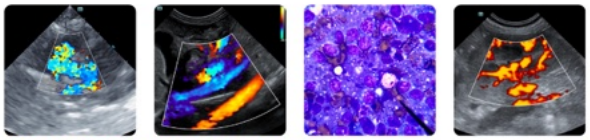

PATIENT

Dez Lamantia

SPECIES

Canine

BREED

Boxer Mix

SEX

Neutered male

AGE

10 years

WEIGHT

67.8 lbs

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY

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PRESENTING CLINICAL SIGNS

History: Chronic skin condition currently on cyclosporine Waxing and waning GI signs with presumed sharp GI pain per O On thyroid tabs Elevated proBNP - echo unremarkable - echo read by Sonopath incase you need to review back.
Abnormal PE/Chem/CBC/UA Results: Mildly low BG

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The **urinary bladder**, trigone, and pelvic urethra presented normal thicknesses and normal tone. The ureters were not visible which is normal. No uroliths or sediment were visualized and anechoic urine was present. No evidence of inflammatory or neoplastic changes was noted. Ureteral papillae were normal.

The **kidneys** revealed normal size and structure, corticomedullary definition and ratio for this age. The cortices presented largely uniform texture with normal echogenic relationship to liver and spleen. Medullary structure differed distinctly from the cortex and no evidence of pelvic dilation was present. The capsules were acceptably uniform without significant irregularities. The left kidney measured 5.05 cm. The right kidney measured 5.32 cm.

Adrenal Glands

The **adrenal glands** were not overtly visualized. However, the regions of the adrenal glands appeared unremarkable.

Spleen

The **spleen** in this patient was mildly enlarged with uniform parenchyma and was folded upon itself. This is a positional variant and is not pathological. There was no evidence of significant disease.

Liver

The **liver** images submitted revealed subjectively normal liver size, contour, and structure. Parenchymal echogenicity was naturally coarse and hypoechoic to the spleen. Vascular and biliary tracts were of normal volume with no evidence of congestion. The gallbladder presented acceptably thin walls with primarily anechoic content. The cystic and common bile ducts were normal. No pathological hepatic lymphadenopathy was evident. No overt structural evidence of inflammatory, infiltrative or regenerative pathology was evident.

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Gastrointestinal

Examination of the **gastrointestinal tract** revealed a stomach and intestine free of stasis, of normal wall thickness, acceptable curvilinear mural detail, and peristaltic activity. Small and large intestine demonstrated normal luminal chyme and stool consistency respectively. No obstructive or overt infiltrative disease was noted. No associated abnormal lymphatic activity was noted.

Pancreas

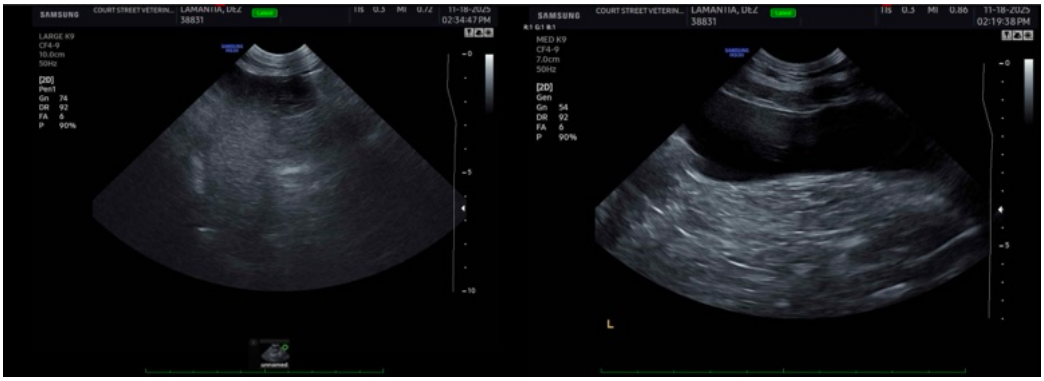
The base and limbs of the **pancreas** were observed to be largely isoechoic to surrounding omental fat. Pancreatic duct and capsular contour were acceptably normal and parenchyma respected normal curvilinear patterns. No overt evidence of active inflammatory or neoplastic disease was noted.

ULTRASONOGRAPHIC FINDINGS

Structurally unremarkable abdomen with mild uniform splenomegaly and splenic fold.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The folded spleen was causing the appearance of splenomegaly on radiographs, yet not pathological. There was no evidence of significant pathology. The cause of low blood glucose is unclear.



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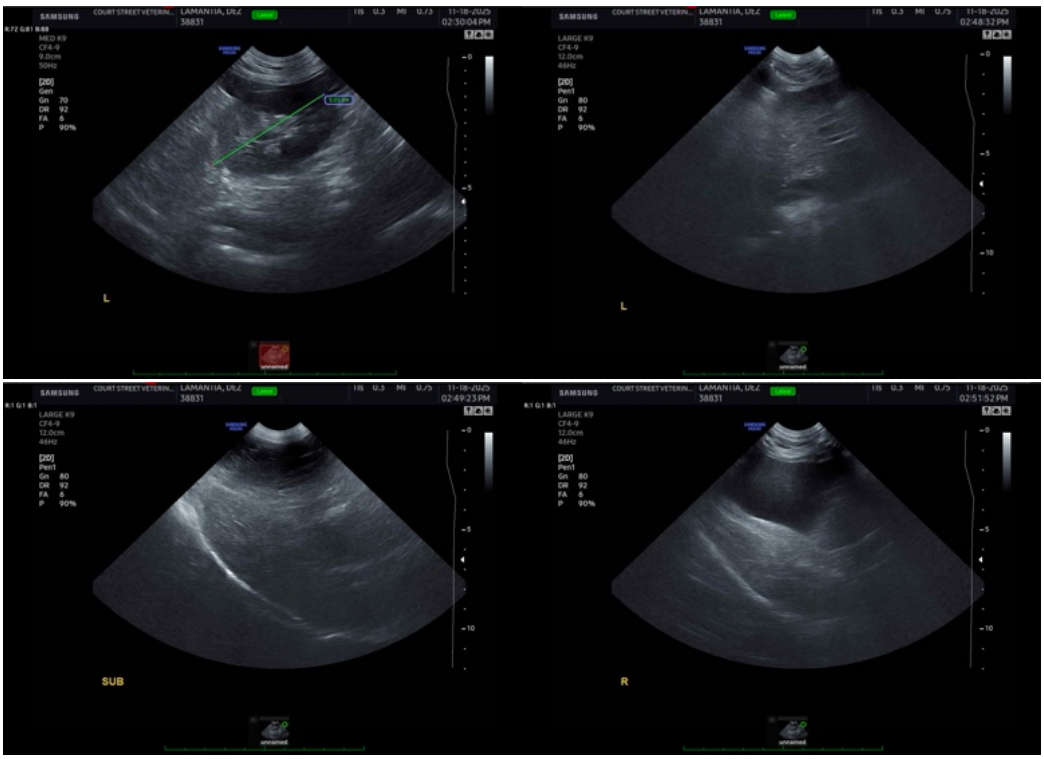

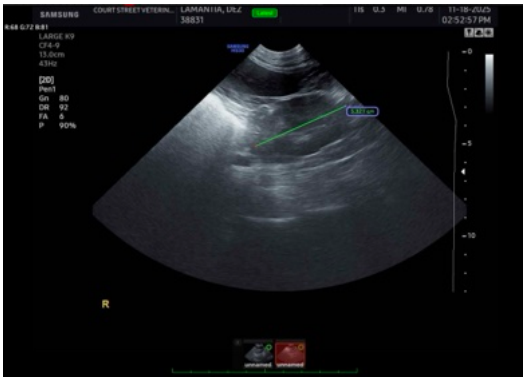

The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

Eric Lindquist, DMV, DABVP (CFM), Cert. IVUSS, CEO of SonoPath.com

info@SonoPath.com

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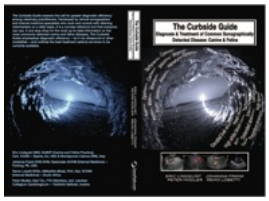
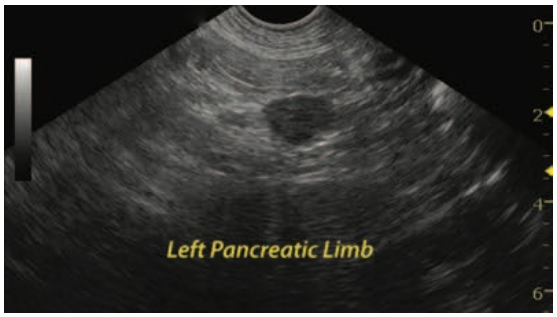


The following is an applicable excerpt from the *Curbside Guide to Diagnosis & Treatment of Sonographic Disease* offered by SonoPath.com Lindquist, Frank, Lobetti, and Modler.

An essential quick guide for every general practitioner and sonographer.

Hypoglycemic Syndrome: Insulinoma and Other

http://www.sonopath.com/Hypoglycemia



Short axis of the left pancreatic limb in a dog with an insulinoma seen as an ovoid hypoechoic mass lesion expanding the pancreatic capsule.

Description: Hypoglycemia can be found incidentally or associated with non-specific clinical signs, such as listlessness and weakness. It is essential to consider the multiple differentials for hypoglycemia in order to avoid a potential hypoglycemic crisis. One must perform a rapid and efficient workup to arrive at a diagnosis and prescribe the proper therapy.

Differentials for hypoglycemia include: laboratory or handling error; sepsis; toxins (e.g. xylitol, ethylene glycol); hunting dog hypoglycemia; Addison's disease; polycythemia; liver failure; poorly regulated diabetes mellitus; and neoplasia (e.g. leiomyosarcoma, hepatic, lymphoma, and insulinoma).

Once other causes of hypoglycemia have been ruled out, one may initiate an investigation into the possibility of insulinoma. Insulinoma is a tumor of the pancreas that originates in the beta cells and leads to the unregulated secretion of insulin and hypoglycemic syndrome. The tumor can be a malignant carcinoma or a more benign form of adenoma. There is, however, controversy regarding the exact histopathology associated with insulinoma types.

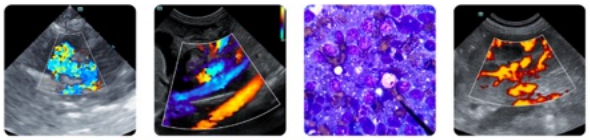
Insulinoma patients are usually middle-aged dogs. Half of all cases present with metastasis to the lymph nodes, liver, and mesentery at the time of diagnosis. There are 3 stages of insulinoma:

Stage 1: Pancreatic localization

Stage 2: Pancreas and lymph nodes with a median survival time (MST) of 1.5 years

Stage 3: Organ metastasis with an MST of 6 months.

Clinical Signs: Neurogylocopenia syndrome results in lethargy, ataxia, collapse, and seizures.



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Catecholamine release from hypoglycemia leads to hunger, behavior changes, and muscle tremors. Postprandial exacerbation of clinical signs can occur.

Diagnostics: When investigating for insulinoma, one should use a fluoride-containing tube (i.e., a grey top tube) to obtain an accurate glucose level. A fasting glucose level below 60 mg/dl is diagnostic for hypoglycemia. Insulinoma is indicated when one observes the Whipple's triad of hypoglycemia, clinical signs consistent are with hypoglycemia, and the latter resolve with the administration of dextrose.

Fasting insulin and glucose ratio: A high normal to elevated insulin level with glucose < 60 mg/dl is diagnostic for insulinoma.

Imaging: Localizing the lesion with staging is best approached by ultrasound. The ability to localize the lesion may be highly operator- and/or machine-dependent given the often small or even microscopic nature of insulinoma, especially early on in the disease. Primary or secondary lesions associated with insulinoma can often be identified with higher resolution sonography. Appropriate ultrasound-guided sampling (FNA or core biopsy) can be performed of any enlarged lymph node or hepatic nodule if a primary pancreatic lesion is not seen.

Computed tomography (CT) with contrast is likely more sensitive than the average sonographer when it comes to assessing insulinoma.

Tumor staging and histopathological characterization in conjunction with the Ki67 biomarker index will yield solid criteria for the prognostic evaluation of insulinoma.

Treatment:

In cases of emergency hypoglycemic crisis, apply corn syrup to the gums. Administer a dextrose bolus (0.5g/kg IV) and maintain 2.5-10% dextrose solution. If cerebral edema occurs, one should administer dexamethasone (2 mg/kg IV) and give mannitol (0.5 mg/kg IV) over a 20-minute period.

One should perform a surgical pancreatectomy if the tumor is localized (i.e., stage 1 insulinoma). Given that the lesion may be difficult to locate with the naked surgical eye or via palpation, the surgical procedure can be enhanced by intraoperative ultrasound.

In cases of stage 2 and 3 insulinoma, administer prednisolone (0.25 mg/kg PO BID). A glucagon IV infusion has also been suggested; it should be infused with saline at 5 mg/kg/min for refractory cases. If prednisone is not adequate, one can supplement with benzothiadiazide diazixide (5 mg/kg PO BID).

Patients should be fed small, frequent portions of a diet high in fat, complex carbohydrates, and protein.

Chemotherapy: In some cases, the use of alloxan (65 mg/ kg IV) has been shown to be helpful.

Conclusion: The largest study of insulinoma patients identified a general MST of 547 days; however, the MST was 785 days for those undergoing pancreatectomy and 1316 days for those that relapsed after surgery and received treatment with prednisone. Other studies have reported an MST of 258 days with pancreatectomy. All of these results indicate that insulinoma is treatable. Using ultrasonography for staging and histopathological characterization is essential in order to determine whether the appropriate treatment should be surgical, medical, or both.

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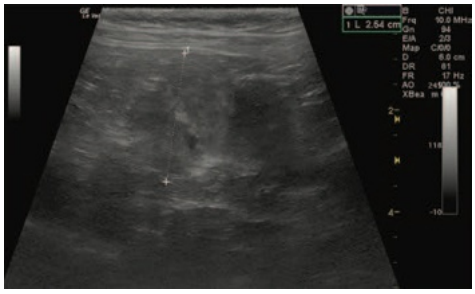
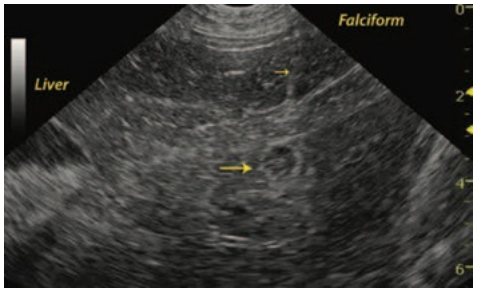
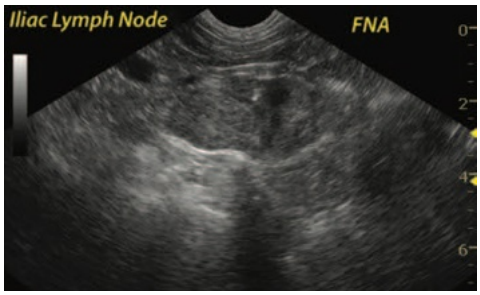


Short axis of the left pancreatic limb in a cat with an insulinoma seen as a complex heterogenous mass lesion expanding the pancreatic capsule (between calipers). Note the mass effect of the tumor displacing the transverse colon caudally.



Subxiphoidal short axis of the liver in a dog with an insulinoma during ultrasound guided sampling of a suspected metastatic lesion.

The needle trajectory is seen as a hyperechoic line (small arrow) approaching the hypoechoic nodule (large arrow) within the liver parenchyma.



Long axis of the medial iliac lymph node during ne needle aspiration in a dog with multifocal metastatic spread of an insulinoma. The echogenic needle tip is seen within the lymph node. The metastatic lymph node is enlarged, rounded, hypoechoic and heterogenous. The primary metastatic loci in insulinoma are the regional lymph nodes (hepatic, pancreaticoduodenal, gastric) and the liver. Hence, metastatic insulinoma lesions in the iliac lymph node in this case was not a typical occurrence.

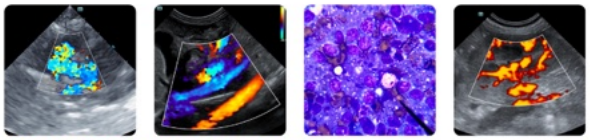
References:

Buishand FO, Kik M, Kirpensteijn J. Evaluation of clinico-pathological criteria and the Ki67 index as prognostic indicators in canine insulinoma. *Vet J* 2010;185:62-67.

Fischer JR, Smith SA, Harkin KR: Glucagon constant-rate infusion: A novel strategy for management of hyperinsulinemic-hypoglycemia crisis in the dog. *J Am Anim Hosp Assoc* 2000;36:27-32.

1.1.Polton GA, White RN, Brearley MJ, et al. Improved survival in a retrospective cohort of 28 dogs with insulinoma. *J Small Anim Pract* 2000;48:151-56.

Steiner JM, Bruyette DS: Canine insulinoma. *Compend Contin Educ Pract Vet* 1996;18:31-36.



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Tobin RL, Nelson RW, Lucroy MD, et al. Outcome of surgical versus medical treatment of dogs with beta cell neoplasia: 39 cases (1990-1997). *JAVMA* 1999;215:226-30.