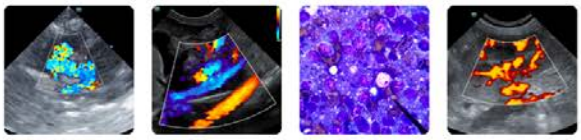


DATE

4/10/26

PATIENT

Fergus Fortier

SPECIES

Canine

BREED

Shih Tzu

SEX

Neutered Male

AGE

12/25/13

WEIGHT

15.9 lbs

INTERPRETED BY

Eric Lindquist, DMV, DABVP, Cert. IVUSS

HOSPITAL NAME

Chadwell Animal Hospital

REFERRING VET

Dr. Mengers

INVOICE

74399

PRESENTING CLINICAL SIGNS

Patient History: Presented 4/7/26 for checking growths, discussing dental. On PE, mod-severe dental calc, multiple papillomas causing irritation and rupturing, uveal cysts, immature cataracts, KCS. Started on optimmune eye drops for KCS and topical animax for papillomas. Pre-dental BW showed elevatd calcium and further elevations of ALP and cholesterol from previous BW conducted in 3/4/2025 and 10/26/23. Recommended fasted iSTAT, rectal exam, and repeat STT

Current Medications: Animax - Apply to ruptured papillomas q12h PRN, tacrolimus - 1 drop OU q12h (switched from optimmune P was initially on due to pharmacy availability)
Labwork Results: Attached, reported as 3/24/26 - CBC/CHEM/T4: Ca 12.7, tp 8.1, glob 4.2, ALP 821
3/4/25 - CBC/CHEM/T4: Ca 11.7, ALP 533, chol 370
10/26/23: ALP 382, Ca 11
Date of Previous IntraPet Ultrasound: No previous.
Sedation: Not required to complete full diagnostic ultrasound.
Stat Report: Not requested.
Imaging Performed by: Stephanie Warga RDCS, RVT.

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The **urinary bladder** itself appeared unremarkable. However, sand was present in the cystourethral junction, proximal urethra and prostatic urethra.

The **kidneys** presented pelvic and corticomedullary calculi. Left kidney measured 3.96 cm. Right kidney measured 4.6 cm.

Adrenal Glands

The **right adrenal gland** was slightly irregular and heterogeneous with a hyperechoic nodule measuring 0.92 cm at the cranial pole. The right adrenal gland measured 2.54 cm x 1.05 cm at the cranial pole and 0.46 cm at the caudal pole.

The **left adrenal gland** presented normal size and contour, measuring 2.15 cm x 0.65 cm at the caudal pole and 0.62 cm at the cranial pole.

Spleen

The **spleen** presented a smooth homogeneous parenchyma hyperechoic to liver and renal cortical parenchyma. The capsule was smooth without noticeable expansion or deviation from within the spleen or adjacent pathology. The splenic vasculature demonstrated normal volume without signs of congestion or thrombosis. No sonographic evidence of acute or chronic inflammatory, neoplastic, or infarctual changes were noted.

Liver

The **liver** images from right and left intercostal as well as subcostal views revealed subjectively normal liver size, contour, and structure. Some age-related parenchymal remodeling was noted but likely not clinically significant at this time. Vascular and biliary tracts were of normal volume and no evidence of congestion was noted. The **gallbladder** was mildly over distended with suspended and dependent debris, yet not to the level of emerging mucocele, yet sludge appears to be mildly excessive. No adjunctive inflammation was noted.

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

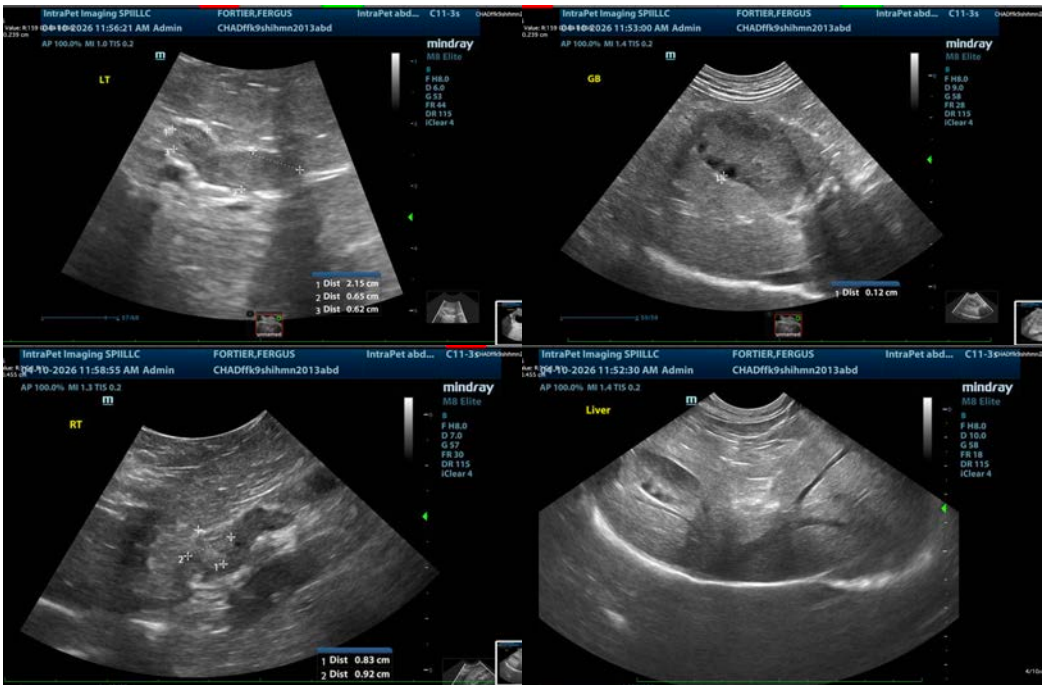
The base and limbs of the **pancreas** were observed to be largely isoechoic to surrounding omental fat. Pancreatic duct and capsular contour were acceptably normal and parenchyma respected normal curvilinear patterns. No overt evidence of active inflammatory or neoplastic disease was noted.

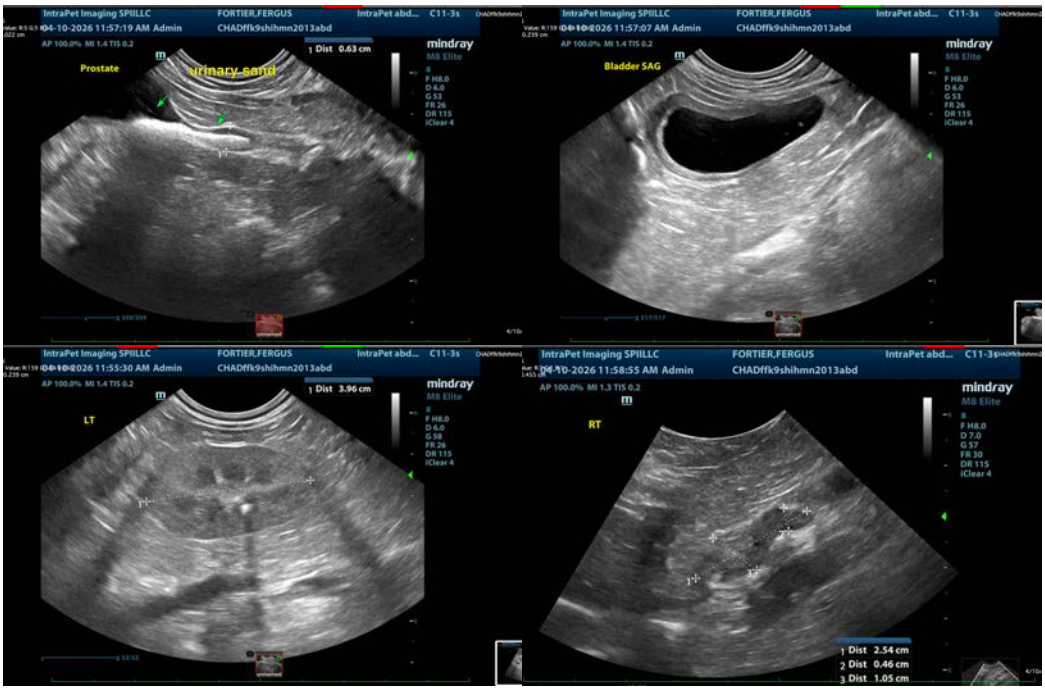
ULTRASONOGRAPHIC FINDINGS

- Bladder, urethral, and renal calculi, likely oxalate given the patient history and echotexture.
- Heterogeneous nodular right adrenal gland.
- Age related hepatic changes.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The cause of hypercalcemia is unclear in this patient. Hypercalcemia panel warranted with potential imaging of the anal glands, cranial mediastinum, and parathyroids. Eventual cystotomy, sand analysis and culture indicated.

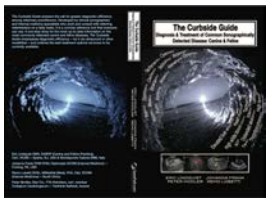




The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

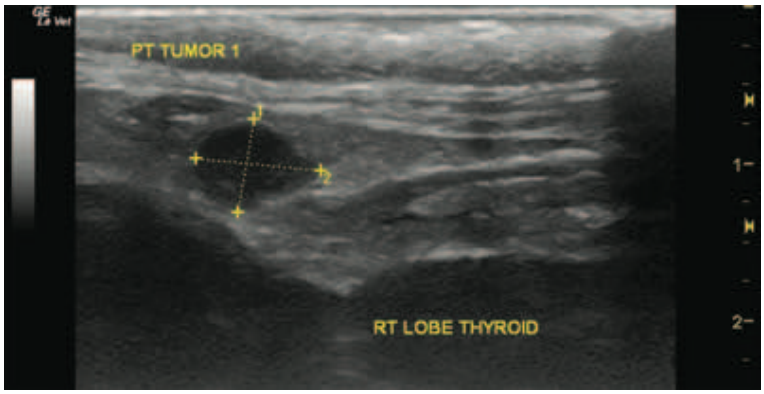
Eric Lindquist, DMV, DABVP(CFM), Cert. IVUSS,
CEO, Owner, Founder -- SonoPath.com
info@SonoPath.com



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.
https://sonopath.com/products/curbside-guide-editing-due-release-12012015

CANINE HYPERCALCEMIA

http://www.sonopath.com/CanineHypercalcemia



Long axis of the right thyroid lobe in a dog with a parathyroid adenoma. The right internal parathyroid gland (between calipers) shows severe uniform enlargement of more than 7mm.

Description: Hypercalcemia is defined as either a persistently elevated total calcium serum (> 12 mg/dl) or ionized calcium (> 1.45 mmol/l) concentration. Clinical signs are often absent with mild hypercalcemia (< 13 mg/dl). In fact, hypercalcemia is often only discovered when serum biochemistry is done for unrelated reasons. Clinical signs are usually mild when the serum calcium concentration is less than 14 mg/dl; however, signs become more readily apparent when the concentration exceeds 15 mg/dl. Life-threatening cardiac arrhythmias can develop when the serum calcium exceeds 18 mg/dl.

Common etiologies of hypercalcemia include humoral hypercalcemia of malignancy (HHM), hypoadrenocorticism, chronic kidney disease (CKD), hypervitaminosis D, and primary hyperparathyroidism. Less common etiologies include bone neoplasia, osteomyelitis, hypertrophic osteodystrophy, granulomatous disease, calcium supplementation, and oral phosphate binders.

Clinical Signs: Common clinical signs include polyuria, polydipsia, lethargy, inappetence, and weakness. With chronic hypercalcemia, calcium oxalate and calcium phosphate uroliths can form, resulting in clinical signs suggestive of lower urinary tract disease. Systemic signs of illness are suggestive of HHM.

Diagnostics: One important etiology of hypercalcemia is laboratory error; therefore, hypercalcemia should always be confirmed before embarking on any further diagnostic evaluation. Results of a CBC, serum biochemistry panel, and urinalysis, in conjunction with a patient history and findings from a physical examination, can often provide enough information to arrive at a diagnosis. The appendicular skeleton, peripheral lymph nodes, abdominal cavity, and rectum should all be carefully palpated for masses, lymphadenopathy, hepatomegaly, splenomegaly, and/or pain in the long bones. The following diagnostic tests

are helpful for identifying an underlying malignancy: thoracic and abdominal radiographs; abdominal ultrasound; cytological evaluation of aspirates of the liver, spleen, lymph nodes, and bone marrow; determination of serum ionized calcium, parathyroid hormone (PTH), and parathyroid hormone-related protein concentration (PTHrP); and ultrasound of the neck. Ascertaining the concentrations of serum ionized calcium, PTH, and PTHrP helps differentiate primary hyperparathyroidism from HHM. The finding of one or more enlarged parathyroid glands upon conducting an ultrasound of the neck supports a diagnosis of primary hyperparathyroidism.

Hypoadrenocorticism-induced hypercalcemia usually occurs in conjunction with hyponatremia, hyperkalemia, and prerenal azotemia. With HHM and primary hyperparathyroidism, serum phosphorus concentration is often in the low to low-normal reference range. If the serum phosphorus concentration is high but kidney function is normal, hypervitaminosis D or osteolysis should be suspected.

It can be difficult to determine whether kidney failure is primary or secondary to hypercalcemia when hyperphosphatemia and hypercalcemia coexist with azotemia. Serum ionized calcium concentrations are typically normal or decreased in cases of renal failure and increased in cases of hypercalcemia caused by other disorders.

Sternal and hilar lymphadenopathy is common with lymphoma-induced hypercalcemia and can be readily identified on thoracic radiographs. In cases of multiple myeloma, discrete lytic lesions in the vertebrae or long bones, hyperproteinemia, proteinuria, and plasma cell infiltration in the bone marrow may be present. Cytological evaluation of the peripheral lymph nodes, bone marrow, and spleen can be helpful in identifying lymphoma.

Increased serum ionized calcium concentrations, detectable serum PTHrP concentrations, and non-detectable serum PTH concentrations are all diagnostic for HHM. Lymphoma is the most common etiology of HHM, but other tumors, such as apocrine gland adenocarcinoma and various carcinomas (e.g. mammary gland, squamous cell, bronchogenic), can all give rise to hypercalcemia. Increased serum ionized calcium, normal to increased serum PTH, and non-detectable PTHrP concentrations are diagnostic of primary hyperparathyroidism.

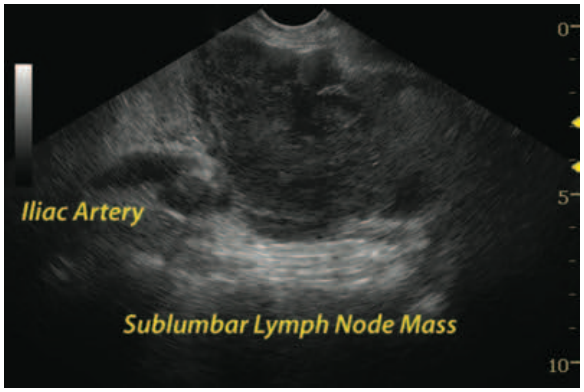
Differentials for Hypercalcemia: "HARD IONS"

Hyperparathyroid
Addison's
Renal
D-toxicity
Idiopathic
Osteolytic
Neoplastic
Spurious

PTH tumor: Elevated total and ionized Ca, low PTHrP, and normal/high PTH. Keeshonds, German Shepherds, and Golden Retrievers are all predisposed.
Addison's disease: Elevated total and normal ionized Ca, elevated BUN, hypoalbuminemia and hyperkalemia.
Renal failure: Elevated to normal total Ca, low ionized Ca, low PTHrP, elevated PTH, azotemia, and low urine specific gravity.
Vitamin D toxicity: Elevated total and ionized Ca, low PTHrP, and normal/low PTH.
Hypercalcemia of malignancy (HHM): Elevated total and ionized Ca, high PTHrP, and low PTH.
Granulomatous disease: Elevated total and ionized Ca, low PTHrP, and low PTH.

Renal failure: Elevated to normal total Ca, low ionized Ca, low PTHrP, elevated PTH, azotemia, and low urine specific gravity.

Treatment: Therapies for hypercalcemia are aimed at correcting the underlying etiology; however, because prolonged hypercalcemia can result in kidney damage, the use of fluid therapy, furosemide, and possibly prednisone is indicated in all cases to reduce serum calcium levels. Suggested dosages include saline (0.9% 120-180 ml/kg day IV), furosemide (1-4 mg/kg PO TID), and prednisone (0.25 mg/kg PO Q24hr).



Long axis of the left hypogastric lymph node in a hypercalcemic dog with lymphoma and hypercalcemia of malignancy. The lymph node is severely enlarged and rounded with a short-to-long-axis ratio > 0.5 indicating malignant in infiltration. The regular echoarchitecture is lost, the hilus is not recognized, lymph node parenchyma is hypoechoic and heterogenous. Also note the mass effect on the external iliac artery. In light of hypercalcemia, lymphadenopathy in this region could also be owing to anal gland adenocarcinoma which can also be imaged sonographically.

References:

Chew DJ, Schenck PA, Jaege JQ. Clinical disorders of hypercalcemia and hypocalcemia in dogs and cats. Proceedings from the American College of Veterinary Internal Medicine, Charlotte, NC, June 4-7, 2003.

Feldman EC. Disorders of the parathyroid glands. In: Ettinger SJ, Feldman EC, ed. *Textbook of Veterinary Internal Medicine, 7th ed.* St. Louis, MO: Saunders Elsevier; 2010:1722-50.

Peterson ME. Hypercalcemia in dogs & cats: differential diagnosis & treatment. Proceedings from the Western Veterinary Conference, Las Vegas, NV, February 19-23, 2012.