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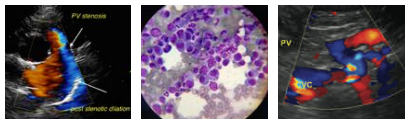


SonoPath
Clinical Sonography & Telecytology
EDUCATIONAL TELECONSULTATION SERVICES™
1-800-838-4268 info@sonopath.com SonoPath.com

**DATE**
10/20/21

**PATIENT**
Bandit Brooks

**SPECIES**
Canine

**BREED**
Terrier Mix

**SEX**
Neutered male

**AGE**
7/25/08

**WEIGHT**

**INTERPRETED BY**
Eric Lindquist, DMV
DABVP, Cert. IVUSS

**HOSPITAL NAME**
Airpark AH

**REFERRING VET**
Dr. Mazzochette

**INVOICE**
92532

## PRESENTING CLINICAL SIGNS

History: Hx of acute onset PU/PD for several weeks. Prior to this: New Lyme positive on screening 4dx on 8/6/21, 1+ proteinuria on UA with 1.042 USG at that time, treated with doxycycline x 4 weeks. Chronic heart murmur, now gr 3-4/6, proBNP WNL 10/4/21.
Current Medications: No current medications.
Lab Results: Mild azotemia with poorly concentrated first AM urine--r/o CKD, pyelonephritis, etc vs pre-renal cause of azotemia with other cause of polyuria. Mild hypercalcemia (12.1)--r/o idiopathic, hyperca of malign, etc. Mildly incr ALKP (318, sl decr from BW 2 months ago)-r/o Cushing's vs benign liver change. ProBNP WNL, which suggests absence of clinically significant heart dz at this time. UA WNL except for decreased USG. Attached separately.
Radiographs: Not provided by the veterinarian.
Date of Previous IntraPet Ultrasound: No previous IntraPet scans.
Sedation: Sedation not required for scan.
Stat Report: STAT report not requested by the veterinarian.

## ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

### *Urinary System*

The **urinary bladder** presented a relatively uniform thickening of the cranioventral and craniodorsal mucosae with micropolypoid mucosal changes without involvement of the submucosae. The urine presented some echogenicity consistent with suspended debris. A trace amount of sand was noted in the bladder. A grouping of which measured 0.4 cm. No evidence of urethral pathology was present. This presentation is most consistent with chronic cystitis. Technically transitional cell carcinoma cannot be ruled out without histopathological review but is not overtly suspected based on this pattern. Cystocentesis and urine culture +/- pathological review of urine cytology would be warranted. No overt calculi were present at this time.

The **kidneys** revealed largely normal size and structure, corticomedullary definition and ratio (cortex 1/3 of medulla) were essentially maintained with some age-related loss of curvilinear patterns regarding the capsule and C/M junction. The cortices presented largely uniform texture with some increased echogenicity expected for this age patient. Medullary structure differed distinctly from that of the cortex and no evidence of pelvic dilation was present. Slight mineralization was noted. The left kidney measured 5.3 cm with mild to moderate degenerative changes.

### *Adrenal Glands*

Both **adrenal glands** were visualized and recognized as having largely normal shape, size, position and acceptable echogenicity for this age group and breed. Some heterogeneity was noted within the adrenal parenchyma without concerning capsular distortion. These changes are likely age related but should be monitored by sonogram should the patient be suspected of having adrenal disease. The right adrenal gland measured 1.03 cm at the cranial pole and 0.65 cm at the caudal pole. The left adrenal gland measured 2.09 x 0.76 cm at the caudal pole and 0.69 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 was noted.

### *Liver*
The left **liver** revealed an anechoic cyst that measured 1.24 cm. The remainder of the liver was unremarkable. The gallbladder presented some dependent debris with essentially normal contour. The cystic and common bile ducts were normal.

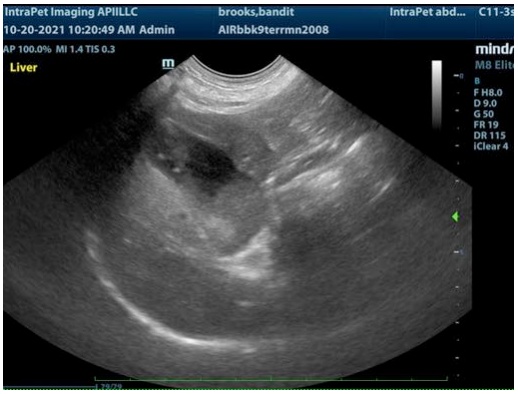
### *Gastrointestinal*
Examination of the **gastrointestinal tract** revealed a stomach and intestine free of stasis, of normal wall thickness, acceptable curvilinear mural detail, and peristaltic activity. Small and large intestine demonstrated normal luminal chyme and stool consistency respectively.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

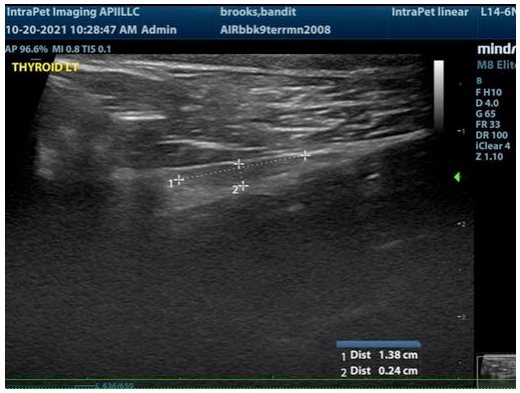
### *Pancreas*
The base and limbs of the **pancreas** were observed to be largely isoechoic to surrounding omental fat. Pancreatic duct and capsular contour were acceptably normal and parenchyma respected normal curvilinear patterns. No overt evidence of active inflammatory or neoplastic disease was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

### *Thyroid*
The right thyroid lobe revealed a hypoechoic, expansive nodule that measured 0.36 cm. The left thyroid lobe was uniform and measured 1.38 x 0.24 cm. The right thyroid lobe measured 1.0 x 0.29 cm.

## ULTRASONOGRAPHIC FINDINGS
Geriatric abdomen.
Minor bladder sand.
Minor renal mineralization.
Parathyroid nodule, consistent with adenoma.

## INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS
If hypercalcemia panel suggests hyperparathyroidism then right parathyroidectomy is recommended. The parathyroid nodule was localized in the mid right lobe of the thyroid.

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IntraPet linear
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THYROID LT
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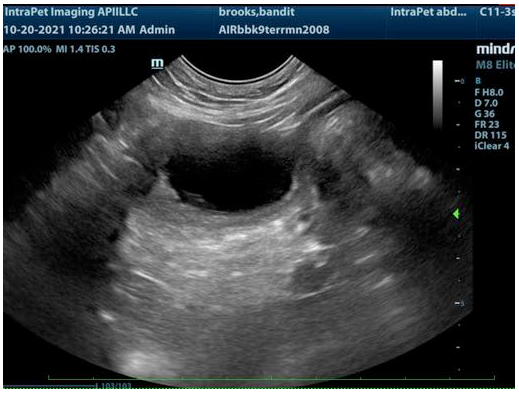
2 Dist 0.24 cm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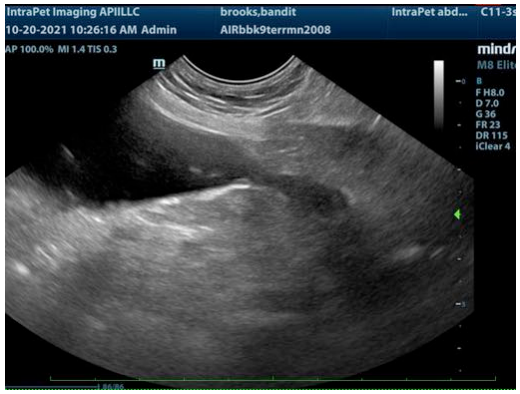
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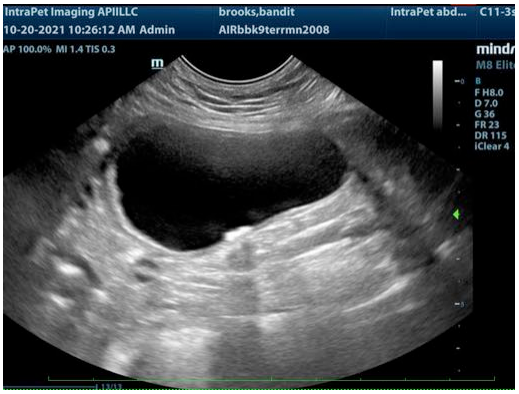
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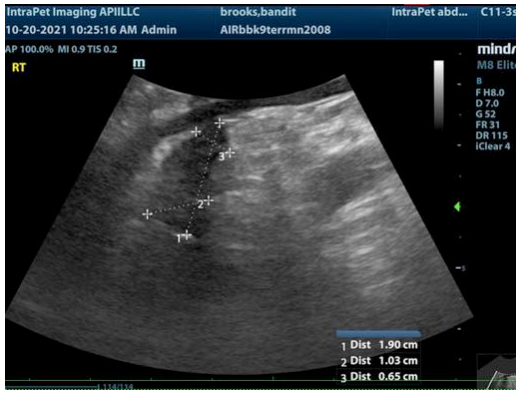
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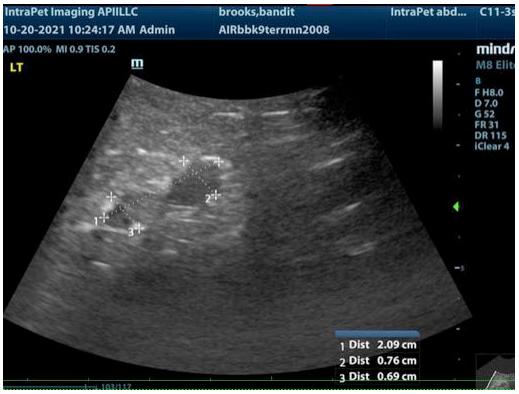
RT
1 Dist 1.90 cm
2 Dist 1.03 cm
3 Dist 0.65 cm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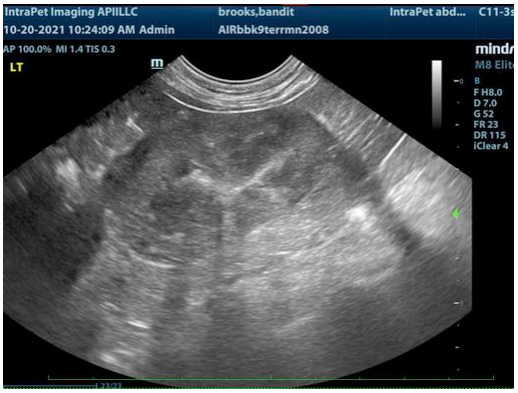
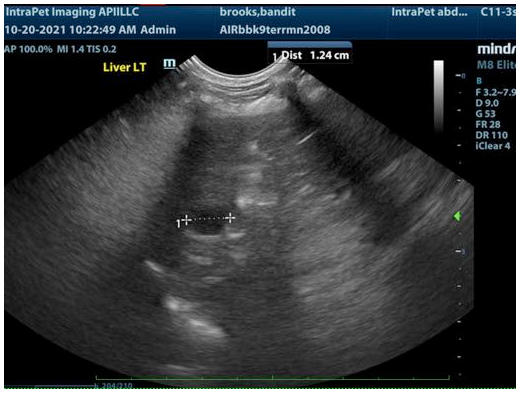
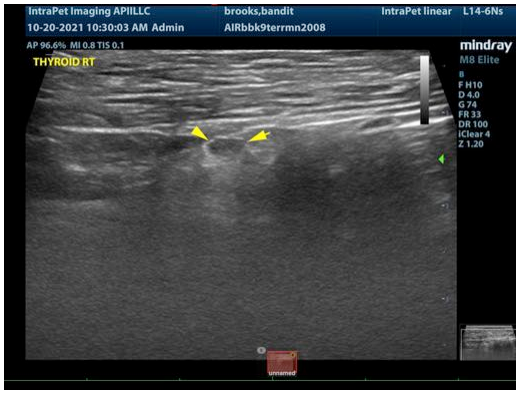
LT
1 Dist 2.09 cm
2 Dist 0.76 cm
3 Dist 0.69 cm



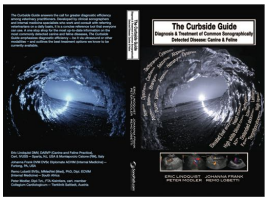




**The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.**

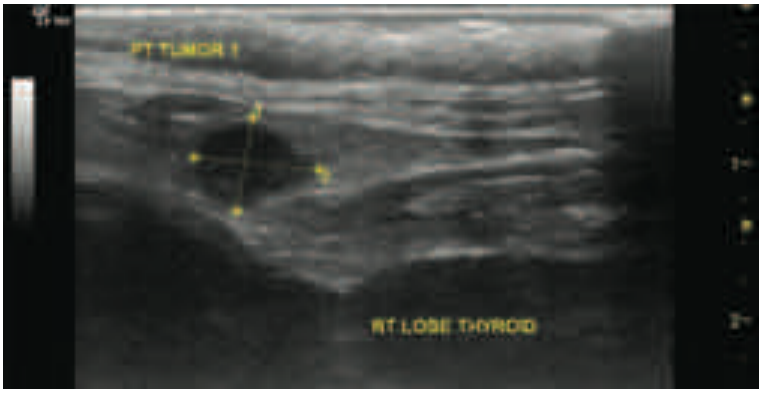
Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance please contact me.

**Eric Lindquist**, DMV, DABVP, Cert. IVUSS, CEO of SonoPath.com
Eric.Lindquist@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.

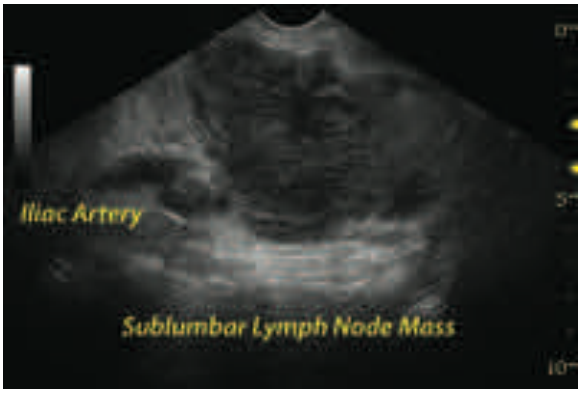
Increased serum ionized calcium concentrations, detectable serum PTHrP concentrations, and non-detectable serum PTH concentrations are all diagnostic for HHM. Lymphoma is the most common etiology of HHM, but other tumors, such as apocrine gland adenocarcinoma and various carcinomas (e.g. mammary gland, 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"**

**H**yperparathyroid
**A**ddison's
**R**enal
**D**-toxicity
**I**diopathic
**O**steolytic
**N**eoplastic
**S**purious

**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.