



**PATIENT**

Walker Franco

**SPECIES**

Canine

**BREED**

PBVG Mixed Breed

**SEX**

Neutered Male

**AGE**

14 Years

**WEIGHT**

30.5 Pounds

**INTERPRETED BY**

Eric Lindquist, DMV,  
DABVP, Cert. IVUSS

**IMAGING PERFORMED BY**

Jenna Walsh, CVT

**HOSPITAL NAME**

VCA Salem AH

**REFERRING VET**

Dr. Giambuzzi

**DATE**

11/4/22

**Invoice**

17978

**PRESENTING CLINICAL SIGNS**

History: suspected hyperparathyroidism and hypercalcemia based on ionized calcium and PTH bloodwork and senior panel done this year. Current Medications Apoquel 16mg 1/2 tab PO SID Primary Question/Differential to Be Answered in This Exam Enlargement/abnormality of thyroid that indicates hyperparathyroidism.

Abnormal PE/Chem/CBC/UA Results: elevated calcium level

**ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN & THYROID**

**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 were noted. Ureteral papillae were normal. The residual prostate was uniform, measuring 0.98 cm.

The **kidneys** revealed largely normal size and structure, corticomedullary definition and ratio (cortex 1/3 of medulla) were essentially maintained with some moderate 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 his age patient. Medullary structure differed distinctly from that of the cortex and no evidence of pelvic dilation was present. An anechoic cyst was noted in the cranial pole of the left kidney, measuring 3.3 cm x 2.3 cm. Some echogenic debris was noted in the left renal cyst. The left kidney measured 6.57 cm.

**Adrenal Glands**

The **right adrenal gland** was visualized and recognized as having normal shape, size, position and echogenicity for this breed. The phrenic vasculature, glandular echogenicity and detail were unremarkable. Capsule, cortex, and medullary definition were normal for this age patient. The right adrenal gland measured 3.02 cm x 0.45 cm at the caudal pole and 0.83 cm at the cranial pole.

The **left adrenal gland** was enlarged, irregular and nodular, measuring 2.96 cm x 1.27 cm at the cranial pole and 0.93 cm at the caudal pole. The left adrenal gland revealed mild irregular capsular expansion without capsular escape or vascular invasion.

**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 moderate age-related parenchymal remodeling was noted but likely not clinically significant at this time. Vascular and biliary tracts were of normal volume and no



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evidence of congestion was noted. No overt evidence of active inflammatory, infiltrative or regenerative pathology was noted but should be paired with current or past LE elevations regarding any clinical significance to this presentation. The hepatic lymph nodes were unremarkable. Echogenic gallbladder wall was noted.

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**Gastrointestinal**

There was some residual chyme and gas was noted in the **stomach**, yet not pathological. This is consistent with end post prandial presentation. Transit of chyme into the small intestine was normal. Curvilinear patterns were maintained throughout the GI tract. No evidence of pathology. 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.

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**Pancreas**

The base and limbs of the **pancreas** were observed to be largely isoechoic to surrounding omental fat. Some mild parenchymal remodeling, however, with mild deviation from curvilinear normalcy was observed. Pancreatic duct and capsular irregularities were present consistent with age related changes. If pain upon imaging (+ Murphy sign) was present or if the patient is focally painful in subxyphoid palpation then low-grade smoldering chronic pancreatitis should be suspected.

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**Thyroid**

The esophagus, trachea and regional tissues were normal.

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The right thyroid lobe was isoechoic to surrounding fat, measuring up to 0.35 cm in width. Normal parathyroids noted, measuring up to 3.0 mm within the body of the right thyroid lobe. However, a focal expansive hypoechoic 0.6 cm x 0.25 cm hypoechoic nodule was noted at the cranial aspect of the right thyroid, suspect parathyroid adenoma. Minor capsular expansion was noted; however, this is low end of expected size for parathyroid adenoma. If hypercalcemia panel suggests primary hyperparathyroidism, then surgical removal of the cranial right parathyroid is indicated.

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The left thyroid lobe measured maximum width of 0.26 cm, uniform and isoechoic to surrounding fat. The largest parathyroid was normal, measuring 0.35 cm in length, embodied within the parenchyma of the thyroid gland. Other smaller normal parathyroids also evident. No pathology associated with capsular expansion or disruption of architecture.

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**ULTRASONOGRAPHIC FINDINGS**

**REFERRING VET**

Dr. Giambuzzi

- Largely geriatric abdomen
- Moderate age-related renal changes with left renal cyst
- Partially full stomach
- Moderate age-related hepatic changes
- Irregular left adrenal gland
- Pronounced right parathyroid nodule, suspect early parathyroid adenoma, to be confirmed by hypercalcemia panel to confirm presence of primary hyperparathyroidism

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**INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS**

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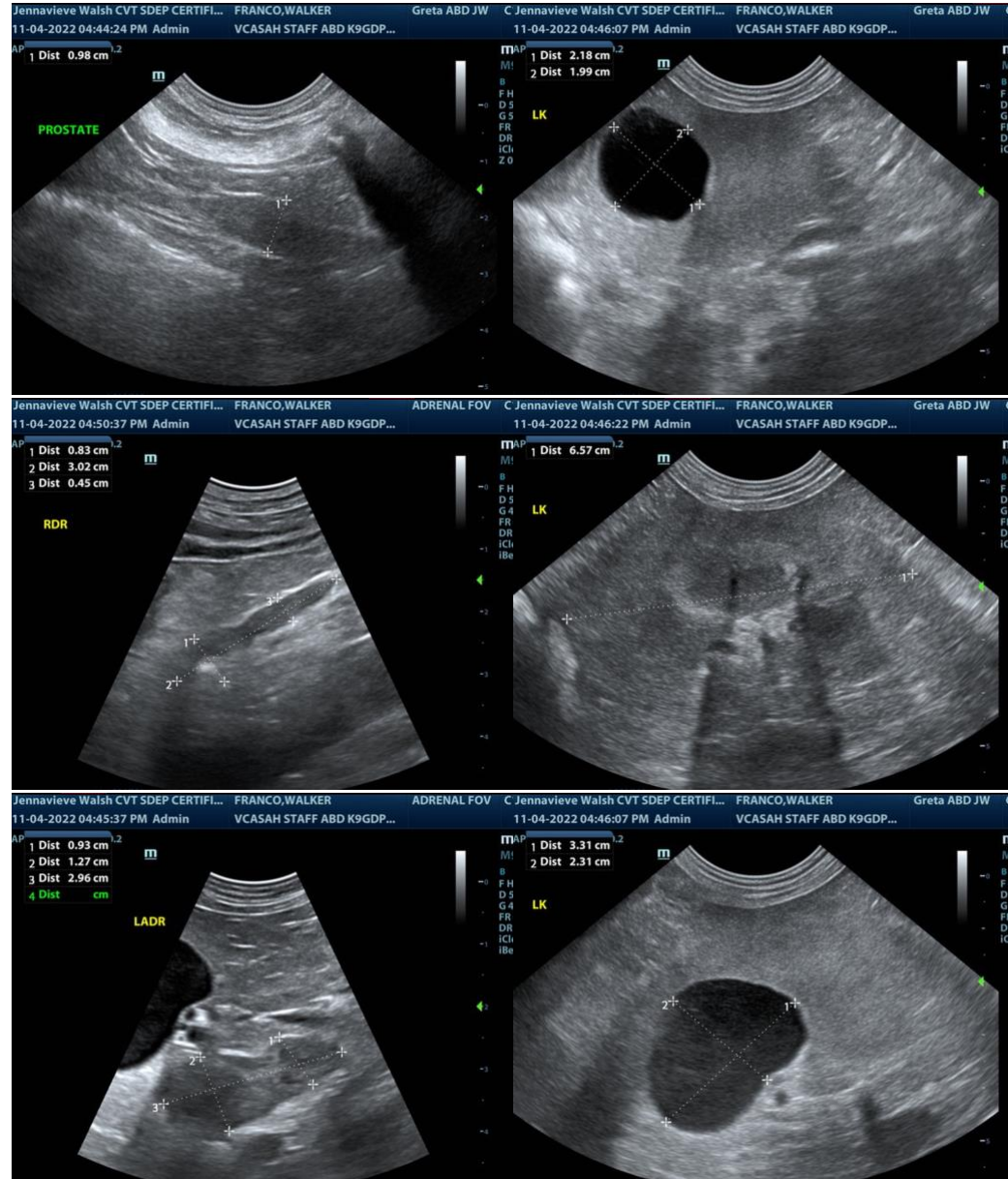
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Left adrenal differentials include hyperplasia (most likely), emerging carcinoma or pheochromocytoma (possible). This should be monitored. If hypertension is an issue, then urine catecholamine is indicated. If the patient appears Cushingoid and urine specific gravity is <1.020, then work up for adrenal dependent Cushings is indicated.





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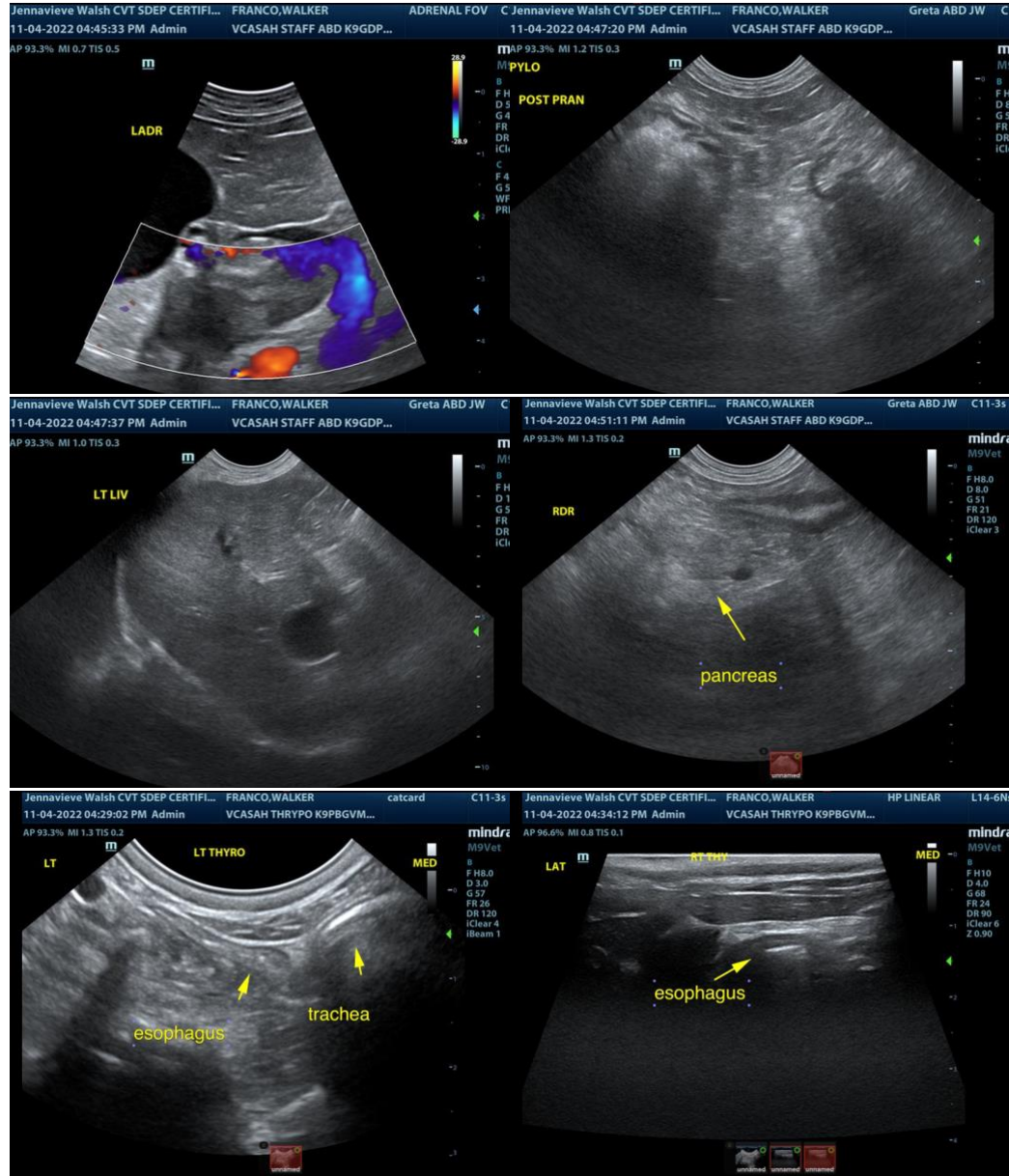
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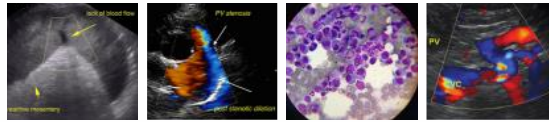
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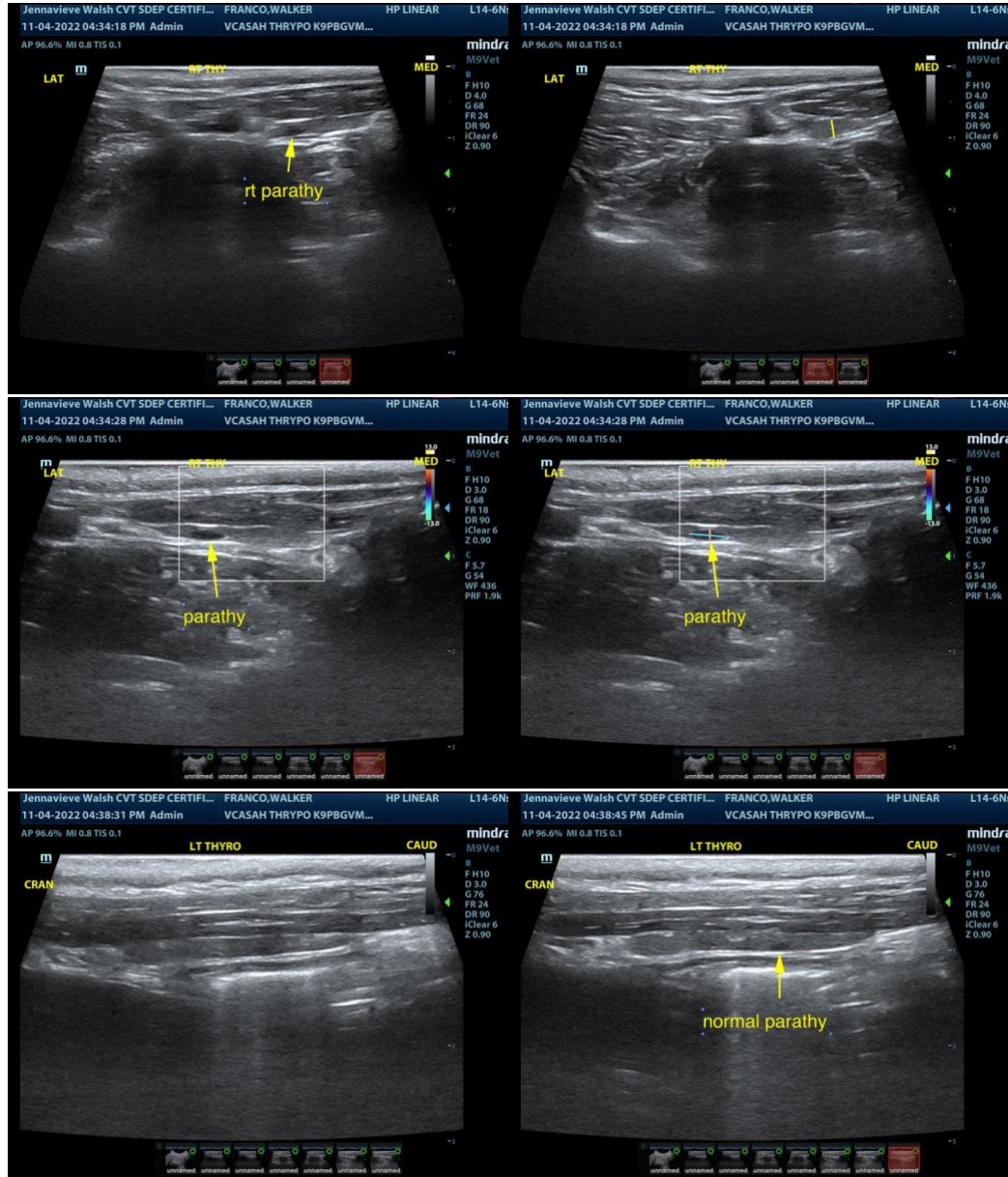
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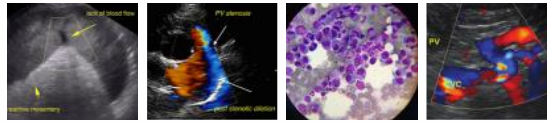
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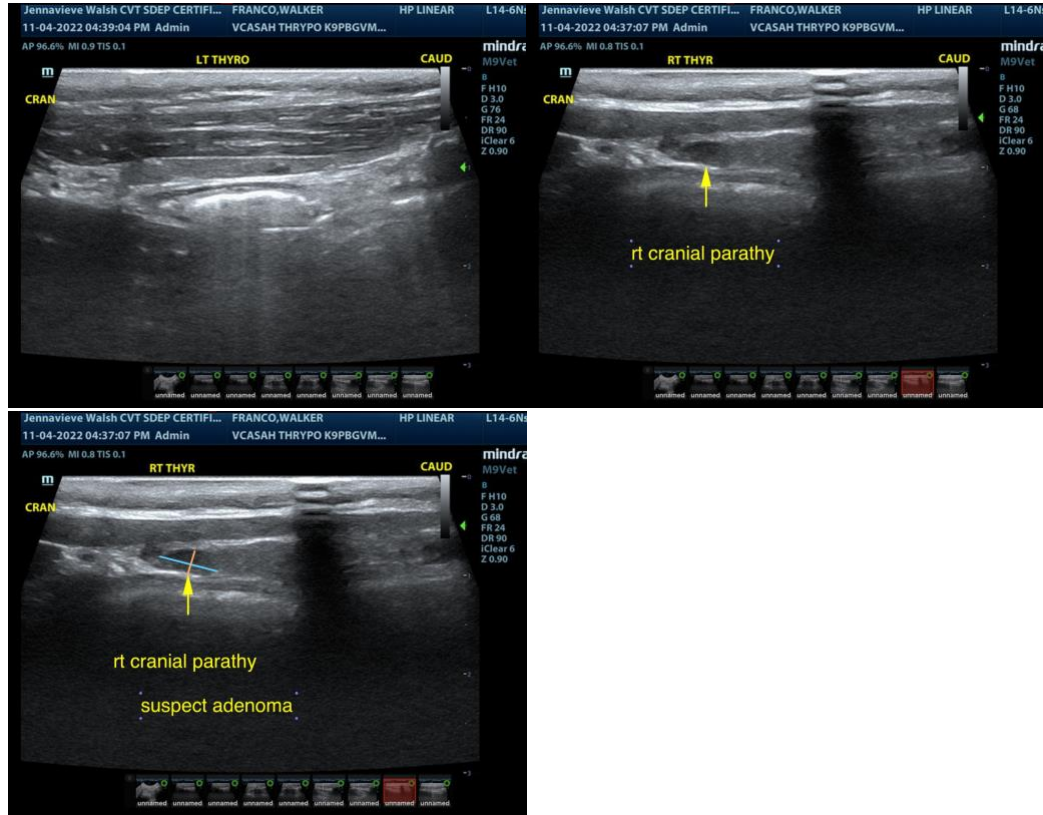
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The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

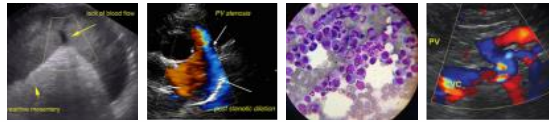
Eric Lindquist, DMV, DABVP, Cert. IVUSS, CEO of SonoPath.com

info@SonoPath.com

**CANINE HYPERCALCEMIA**

<http://www.sonopath.com/CanineHypercalcemia>

**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



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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.

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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.

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**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.

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**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.

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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.

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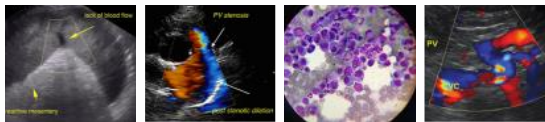
It can be difficult to determine whether kidney failure is primary or secondary to hypercalcemia when hyperphosphatemia and hypercalcemia coexist with azotemia. Serum ionized calcium concentrations are typically normal or decreased in cases of renal failure and increased in cases of hypercalcemia caused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

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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

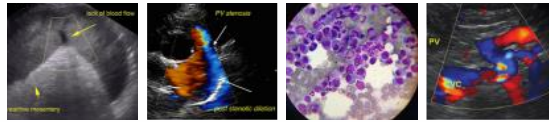
**PTH tumor:** Elevated total and ionized Ca, low PTHrP, and normal/high PTH. Keeshonds, German Shepherds, and Golden Retrievers are all predisposed.

**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.



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**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).

**References:**

Chew DJ, Schenck PA, Jaeger 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.