



PATIENT

Scully Rinkerman

PRESENTING CLINICAL SIGNS

History: alp 1136 ,elevated ;ipase and calcium ionized ca + ua pending pe--pot belly appearance r/o Cushing dz vs other

SPECIES

Canine

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

BREED

Beagle

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 pelvic urethra was imaged 3.0 cm beyond the cystourethral junction.

SEX

Male

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 5.0 cm. The right kidney measured 5.6 cm.

AGE

13 Years

Adrenal Glands

Both **adrenal glands** were 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 0.86 cm at the cranial pole and 0.53 cm at the caudal pole. The left adrenal gland measured 0.7 cm at the caudal pole and 0.66 cm at the cranial pole.

WEIGHT

25.5 Pounds

INTERPRETED BY

Eric Lindquist, DMV
DABVP, Cert. IVUSS

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.

IMAGING PERFORMED BY

Dr. Barnea

Liver

HOSPITAL NAME

Tenaflly Vet Center

The **liver** was uniformly swollen with minor, excessive gallbladder debris and over distension with dependent and suspended bile without evidence of overt mucocele formation. However, excessive sludge was present. The liver presented coarse architecture with mildly increased portal markings and subtle, mixed echogenic changes. This is a mild change, consistent with vacuolar hepatopathy and some level of remodeling and history of inflammatory component. There was no overt suspicion of neoplasia.

REFERRING VET

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Gastrointestinal

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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.

DATE

3/15/23

Pancreas



PATIENT

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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.

SPECIES

Canine

ULTRASONOGRAPHIC FINDINGS

- Benign hepatopathy
- Age-related renal changes
- Structurally unremarkable abdomen otherwise

BREED

Beagle

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

FNA of the liver could be considered for further definition. No overt cause of the hypercalcemia. Anal gland imaging, spinal and chest radiographs are indicated, to assess the cranial mediastinum and lung fields +/- thyroid/parathyroid imaging.

SEX

Male

AGE

13 Years

WEIGHT

25.5 Pounds

INTERPRETED BY

Eric Lindquist, DMV
DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Dr. Barnea

HOSPITAL NAME

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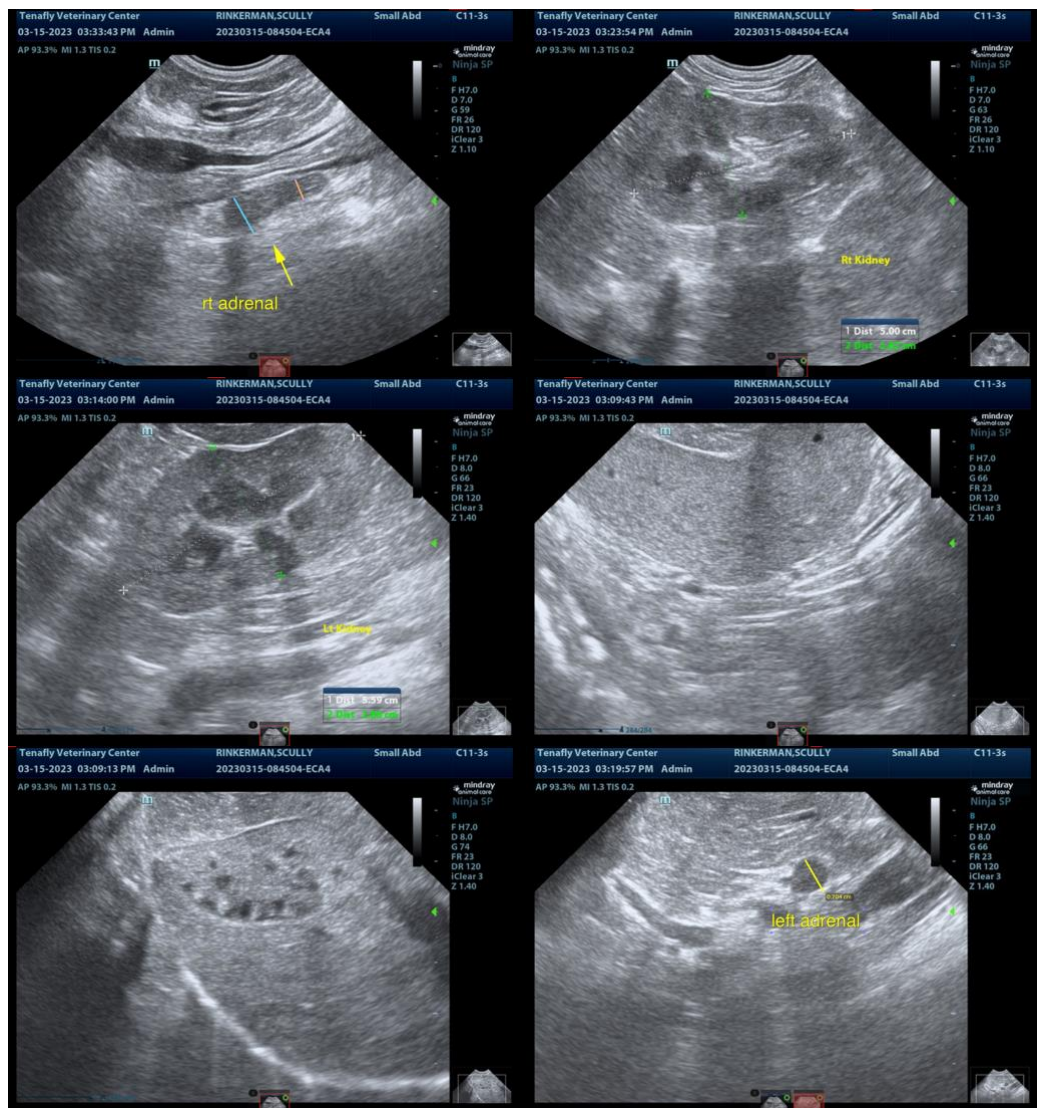
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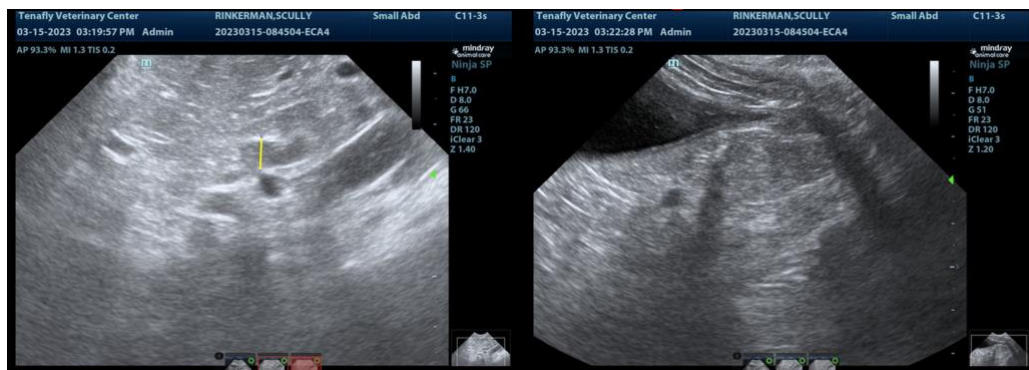
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The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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 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.



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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.

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.

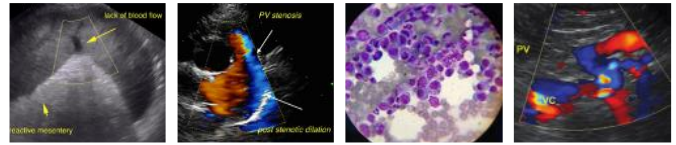
It can be difficult to determine whether kidney failure is primary or secondary to hypercalcemia when hyperphosphatemia and hypercalcemia coexist with azotemia. Serum ionized calcium concentrations are typically normal or decreased in cases of renal failure and increased in cases of hypercalcemia caused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



PATIENT	Addison's
Scully Rinkerman	Renal
SPECIES	D-toxicity
Canine	Idiopathic
BREED	Osteolytic
Beagle	Neoplastic
SEX	Spurious
Male	PTH tumor: Elevated total and ionized Ca, low PTHrP, and normal/high PTH. Keeshonds, German Shepherds, and Golden Retrievers are all predisposed.
AGE	Addison's disease: Elevated total and normal ionized Ca, elevated BUN, hypoalbuminemia and hyperkalemia.
13 Years	Renal failure: Elevated to normal total Ca, low ionized Ca, low PTHrP, elevated PTH, azotemia, and low urine specific gravity.
WEIGHT	Vitamin D toxicity: Elevated total and ionized Ca, low PTHrP, and normal/low PTH.
25.5 Pounds	Hypercalcemia of malignancy (HHM): Elevated total and ionized Ca, high PTHrP, and low PTH.
INTERPRETED BY	Granulomatous disease: Elevated total and ionized Ca, low PTHrP, and low PTH.
Eric Lindquist, DMV DABVP, Cert. IVUSS	Renal failure: Elevated to normal total Ca, low ionized Ca, low PTHrP, elevated PTH, azotemia, and low urine specific gravity.
IMAGING PERFORMED BY	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).
Dr. Barnea	
HOSPITAL NAME	
Tenaflly Vet Center	
REFERRING VET	References:
Dr. Barnea	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.
INVOICE	Feldman EC. Disorders of the parathyroid glands. In: Ettinger SJ, Feldman EC, ed. <i>Textbook of Veterinary Internal Medicine, 7th ed.</i> St. Louis, MO: Saunders Elsevier; 2010:1722-50.
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Peterson ME. Hypercalcemia in dogs & cats: differential diagnosis & treatment. Proceedings from the Western Veterinary Conference, Las Vegas, NV, February 19-23, 2012.

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