



PATIENT

Hercules Conway

SPECIES

Canine

BREED

Mastiff

SEX

Neutered Male

AGE

10 Years

WEIGHT

55 kg

INTERPRETED BY

Eric Lindquist, DMV
DABVP, Cert. IVUSS

**IMAGING
PERFORMED BY**

Erin Wicks

HOSPITAL NAME

Shores VEC

REFERRING VET

Dr. Lupole

INVOICE

13880

DATE

2/11/22

PRESENTING CLINICAL SIGNS

History: Presented at our hospital for declining. P was diagnosed with kidney disease this past Saturday after decreased appetite for about a week, and is no longer eating. P vomited once yesterday and had loose stool once last week. O also noticed a bump on the head that does not seem to bother P. O also reported increased urination. Previous Health Concerns: ear infections Current Medications: cefpodoxime, omega III

Abnormal PE/Chem/CBC/UA Results: rDVM bloodwork: SDMA 26 H, Total T4 1.3 N Chemistry: Calcium 15.2 H, ALT 188 H, GGT 17 H CBC: Lymph 0.81 L, Hgb 19.8 H, HCT 55.7% N EPOC: Ionized Ca 1.89 H Urinalysis: USG 1.016, no crystals, bacteria noted. Waxy casts noted. 1-3 RBC cast/hpf. Trace protein present. Lepto witness test: negative Flex 4: negative x 4 L anal glad, concerned for thickening vs potential mass.

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 were noted. Ureteral papillae were normal. The pelvic urethra was imaged 2.0 cm beyond the cystourethral junction. Iliac trifurcation was unremarkable.

The **kidneys** revealed largely normal size and structure, corticomedullary definition and ratio (cortex 1/3 of medulla) were essentially maintained with some minor 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. The left kidney measured 8.28 cm. The right kidney measured 8.27 cm.

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 left adrenal gland measured 3.9 cm x 0.49 cm at the cranial pole and 0.47 cm at the caudal pole. The right adrenal gland measured 1.0 cm in width.

Spleen

The **spleen** was uniformly enlarged with relatively uniform parenchyma without evidence of masses. The capsule was mildly swollen. This is most consistent with minor hypersplenism and reactive hyperplasia deriving from splenic white or red pulp. However, early infiltrative disease, such as lymphoma or mast cell neoplasia can, at times, present in this manner but not suspected. 25g US-guided FNA would be best in order to ensure only reactive hyperplasia is present. If clinical signs fit with potential neoplasia or mast cell disease, then Benadryl injection (1 mg/pound IM) 15 minutes prior to FNA would be recommended. Caudal folding of the spleen was noted.

Liver



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The **liver** was swollen and irregular with increased portal markings. The gallbladder and common bile duct were unremarkable, other than minor gallbladder wall thickening. Hepatic lymph nodes were mildly enlarged and reactive.

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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.

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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.

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ULTRASONOGRAPHIC FINDINGS

AGE

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- Swollen irregular liver with increased portal markings
- Hepatic lymphadenopathy
- Minor splenic enlargement
- Age-related renal changes

WEIGHT

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INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

I recommend screening FNA of the spleen and liver, assessing for possibility of lymphoma. Cranial mediastinal imaging/radiographs, anal gland palpation +/- parathyroid imaging all indicated. However, the liver is concerning for either chronic inflammatory hepatopathy or emerging hepatic lymphoma. Core biopsy would be ideal. Bile acid profile indicated.

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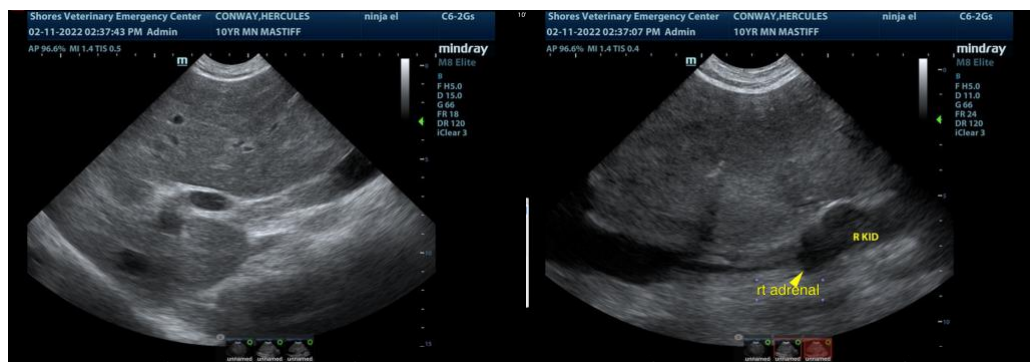
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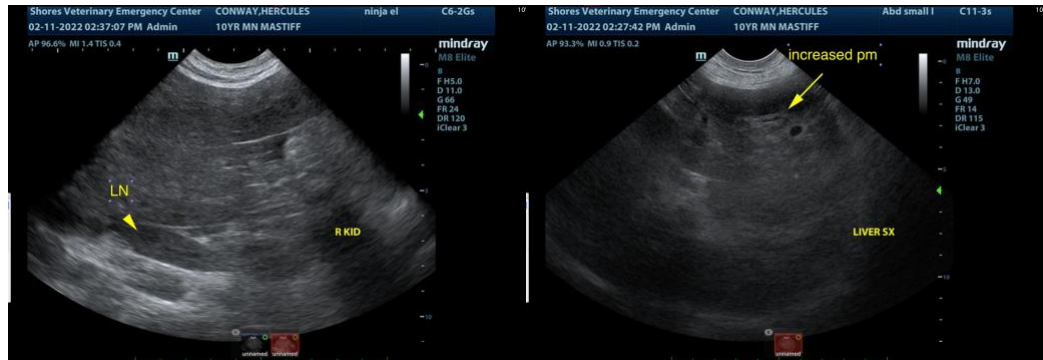
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The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.



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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.

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CANINE HYPERCALCEMIA

BREED

Mastiff

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

SEX

Neutered Male

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.

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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.

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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.

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Differentials for Hypercalcemia: "HARD IONS"

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Hyperparathyroid

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Addison's

Renal

D-toxicity

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PTH tumor: Elevated total and ionized Ca, low PTHrP, and normal/high PTH. Keeshonds, German



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Shepherds, and Golden Retrievers are all predisposed.

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Addison's disease: Elevated total and normal ionized Ca, elevated BUN, hypoalbuminemia and hyperkalemia.

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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.

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Hypercalcemia of malignancy (HMM): Elevated total and ionized Ca, high PTHrP, and low PTH.

Granulomatous disease: Elevated total and ionized Ca, low PTHrP, and low PTH.

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Neutered Male

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

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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).

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References:

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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.

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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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