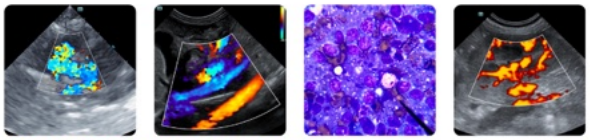

PATIENT

Roxy Torres

SPECIES

Canine

BREED

Boxer Mix

SEX

Spayed female

AGE

9 years

WEIGHT

44 lbs

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Matt Heinlein

HOSPITAL NAME

TLC AH

REFERRING VET

Dr. Castorena

INVOICE

71010

DATE

1/27/26

PRESENTING CLINICAL SIGNS

- 12/17/25 P referred for continued care from urgent care facility after gastrotomy to remove rock foreign body. Hypercalcemia and azotemia noted on pre-operative blood work.
- Hypercalcemia malignancy panel inconclusive for possible hyperparathyroidism
- IM consult recommend thyroid ultrasound imaging
- 01/08/2026 Hypercalcemia of malignancy panel: iCa 1.63 (1.25-1.45) Parathyroid hormone 5.00 (1.10 - 10.6) Parathyroid Related Protein (0.0 - 1.0) 01/08/2026 istat: Na: 144 K:5.0 (3.4-4.9) Cl: 120 TCO2: 18 BUN: 36 (10-25) (increased but improving slowly) Crea: 1.7 (0.5-1.3) (increased but improving slowly) Glu: 107 iCa: 1.71 (1.12-1.40) Increased AnGap: 12 Hct 49 Hb*:16.7 12/26/2026 standard IH BW: CBC WNL Chemistry CREA 1.9 (0.5-1.8) Was 3.0 improving BUN 53 (7-27) was 75 improving Calcium 12.2 (7.9-12.0) was 12.2 , staying consistent TP, ALB, GLOB now all WNL ALP >2000 (historic per owner)

ULTRASONOGRAPHIC EXAMINATION OF THE THYROID

Four videos were submitted in this patient. The left thyroid lobe revealed three nodules, one was hypoechoic nodule and measured 0.8 cm, the second nodule measured 1.1 cm and the third measured 0.33 cm. All three nodules appeared to be encapsulated within the thyroid lobe. The right thyroid lobe revealed a 0.35 cm prominent nodule, likely normal parathyroid. This appears to be fairly normal or upper limits of normal. Regional visible tissues such as the carotid, trachea, esophagus and salivary glands all appear to be normal.

ULTRASONOGRAPHIC FINDINGS

Coalescing left thyroid nodules, strongly consistent with parathyroid adenomas.

Prominent nodule in the right, likely normal parathyroid.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

I recommend surgical removal of the left thyroid lobe and/or ultrasound-guided FNA of the nodular changes for further definition to confirm suspicion of parathyroid adenoma. However, carcinoma cannot be ruled out even though not overtly suspected. No extension into regional tissue is noted. This appears to be surgically resectable.

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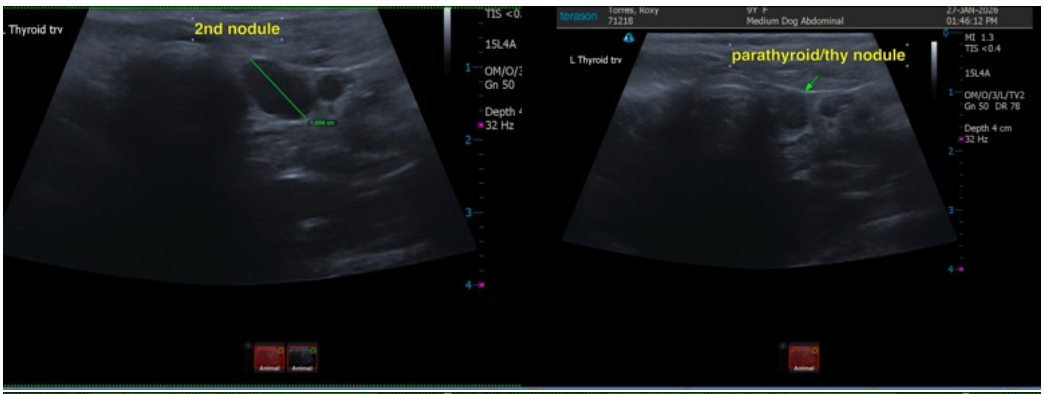
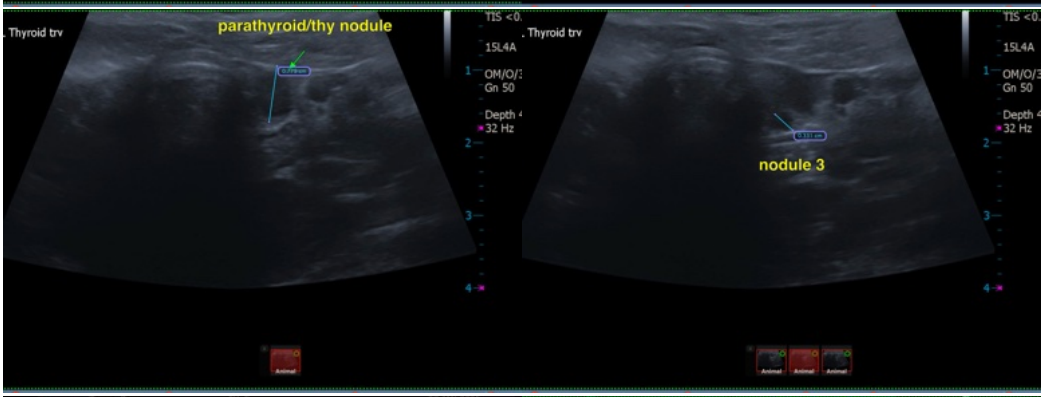


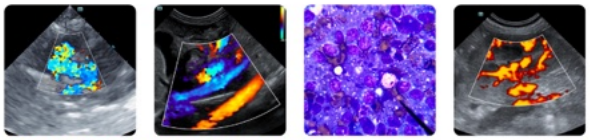


The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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 of SonoPath.com

info@SonoPath.com



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CANINE HYPERCALCEMIA

http://www.sonopath.com/CanineHypercalcemia

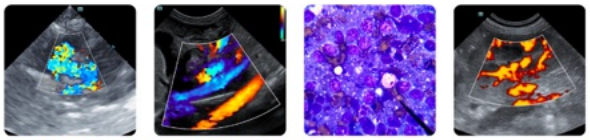
Description: Hypercalcemia is defined as either a persistently elevated total calcium serum (> 12 mg/dl) or ionized calcium (> 1.45 mmol/l) concentration. Clinical signs are often absent with mild hypercalcemia (< 13 mg/dl). In fact, hypercalcemia is often only discovered when serum biochemistry is done 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.

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



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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

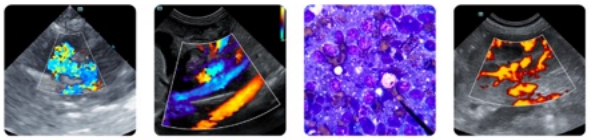
D-toxicity

Idiopathic

Osteolytic

Neoplastic

Spurious



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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).

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.