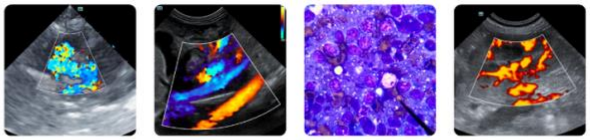

PATIENT

Rooney Schuster

SPECIES

Canine

BREED

Long Haired Dachshund

SEX

Neutered Male

AGE

11 Years

WEIGHT

20.2

INTERPRETED BY

Eric Lindquist, DMV, DABVP (CFM), Cert. IVUSS

IMAGING PERFORMED BY

Brian T. Klug, CVT

HOSPITAL NAME

Ultra Veterinary Sonography

REFERRING VET

Dr. Harminder Singh

INVOICE

36564

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4/12/26

PRESENTING CLINICAL SIGNS

History: PU/PD, Bloodwork recently showed an elevated Calcium and PSL

Abnormal PE/Chem/CBC/UA Results: SuperChem , CBC, T4 and UA on 3/2/26 showed an elevated Calcium, PSL and glucose in urine rest WNL. MSU blood on 3/24/26 showed elevated parathyroid hormone. Previous blood work (5/6/25) showed elevated ALP, B/C ratio, Glucose, amylase and PSL rest WNL

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.

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 6.0 cm. The right kidney measured 5.3 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 0.76 cm at the cranial pole and 0.46 cm at the caudal pole.

The **left adrenal gland** revealed slight irregularity at the caudal pole, to monitor. Slight enhanced fat was noted around the caudal pole of the left adrenal gland. The left adrenal gland measured 0.84 cm at the caudal pole and 0.62 cm at the cranial pole.

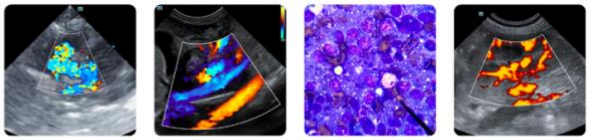
Spleen

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 were noted. The spleen was folded upon itself.

Liver

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 consistent with vacuolar hepatopathy and some level of remodeling and history of inflammatory component. There was no overt suspicion of neoplasia. This is a mild change. Occasional hyperechoic lipid plaques were noted.

Gastrointestinal



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Minor retention of ingesta was noted in the **stomach**. The small intestine and colon were unremarkable.

Pancreas

Both left and right limbs of the **pancreas** revealed remodeling. No evidence of active inflammation was noted.

Free Abdomen

A colic **lymph node** was slightly enlarged and rounded, measuring 0.7 cm.

Other

Both **thyroid** lobes were imaged in this patient with visible parathyroids; the largest of which was the right cranial parathyroid, measuring 0.32 cm, which is the upper limits of normal for parathyroid size. The remainder of the visible parathyroids measured up to 2.0 mm. The thyroid lobes were uniform, no evidence of pathology, measuring approximately 5.0 mm each. The carotid artery, esophagus, trachea, and regional tissues were all normal.

ULTRASONOGRAPHIC FINDINGS

- Slight irregular, nodular, expansive left adrenal gland- differentials include hyperplasia, emerging carcinoma, pheochromocytoma possible.
- Benign hepatopathy
- Slightly enlarged colic lymph node
- Pancreatic remodeling
- Retention of ingesta in the stomach
- Prominent right cranial parathyroid, upper limits of normal. This may represent emerging parathyroid adenoma. No other evidence of potential lesions.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

This is a largely stable abdomen; however, I'm most concerned about the potential emerging pathology in the left adrenal gland and the pancreatic remodeling. No evidence of primary disease related to hypercalcemia in this patient.

Serial blood pressure measurements are recommended in this patient. If hypertension is an issue metanephrine level is recommended. If the patient appears Cushingoid and urine specific gravity is less than 1.020 then work-up for adrenal dependent Cushing's is indicated. Recheck sonogram in one month of the left adrenal gland is indicated, earlier if clinical signs develop.

If primary hyperparathyroidism is suspected, two options are a proactive removal of the right cranial parathyroid or recheck sonogram in 3-4 weeks to assess if this or other lesions emerge, as typically more prominent parathyroid adenomas are >4.0 mm, however, the right cranial parathyroid may represent an emerging adenoma.

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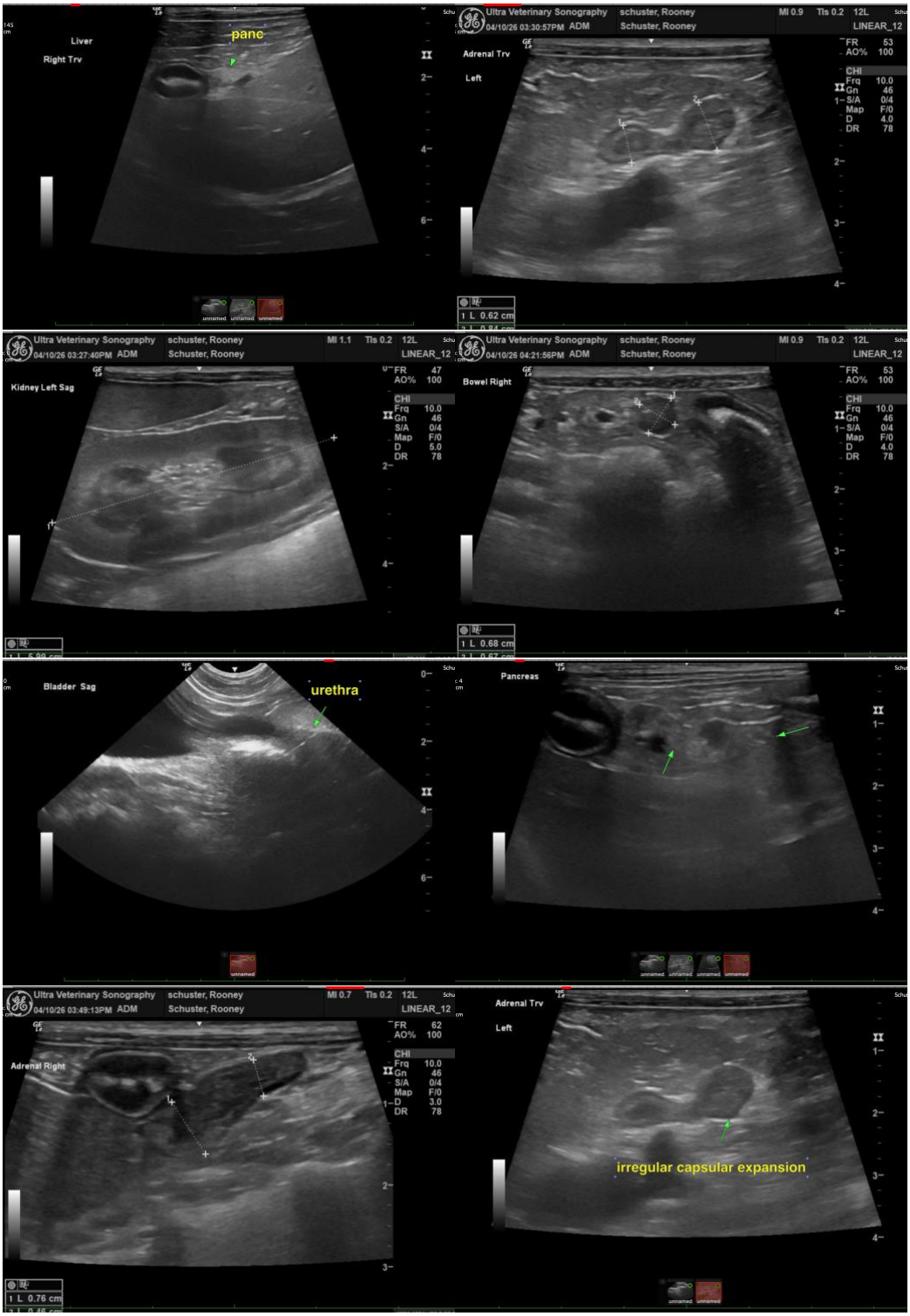

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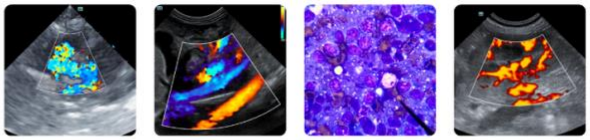

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Educational Teleconsultation Services™

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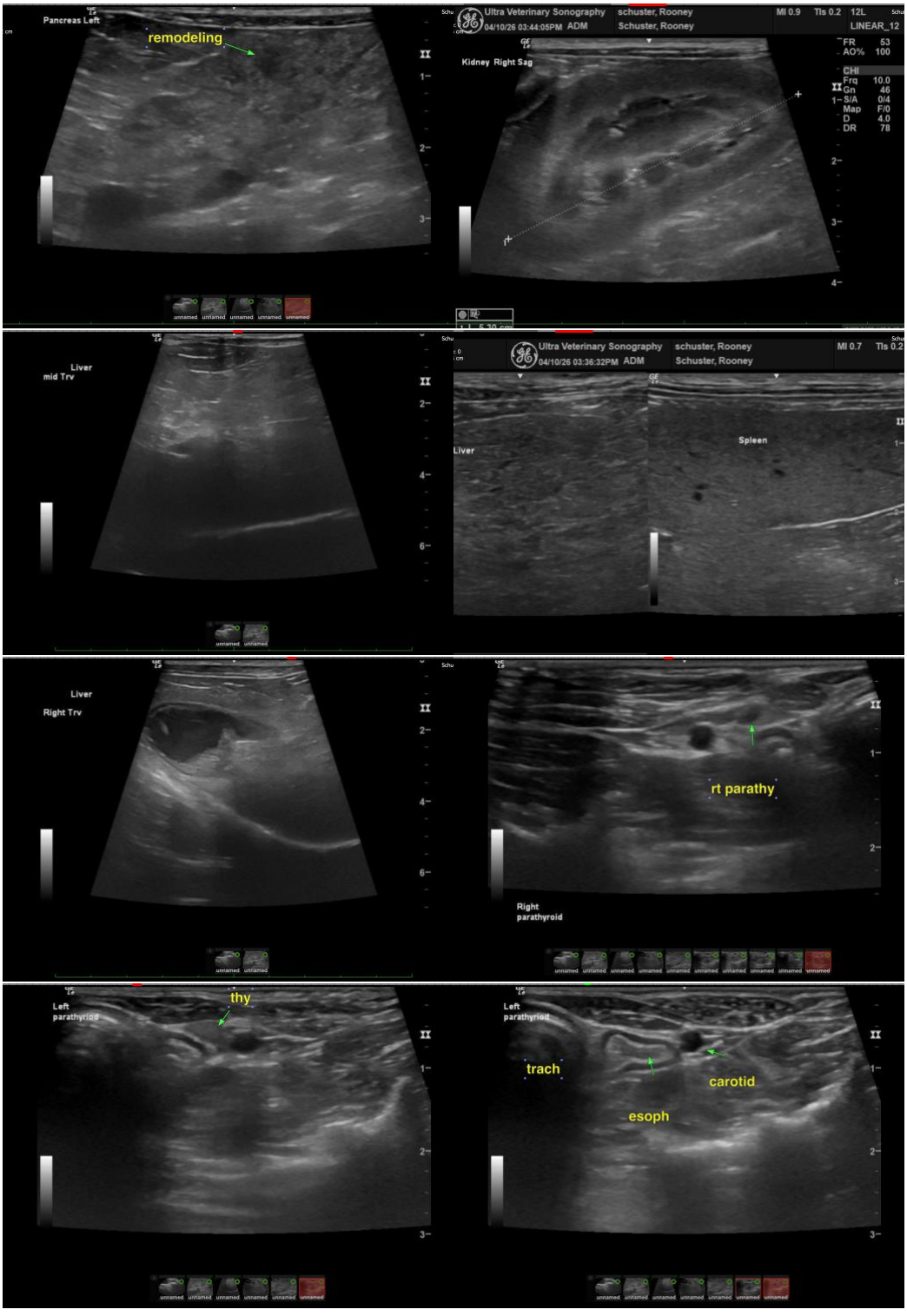

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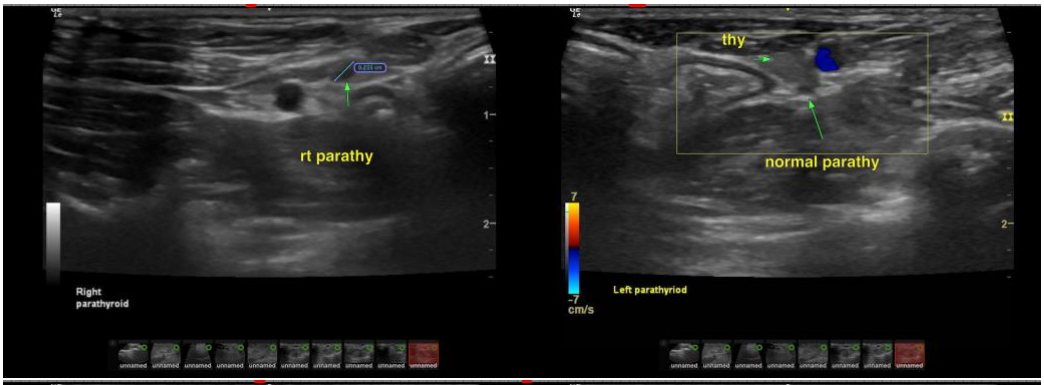
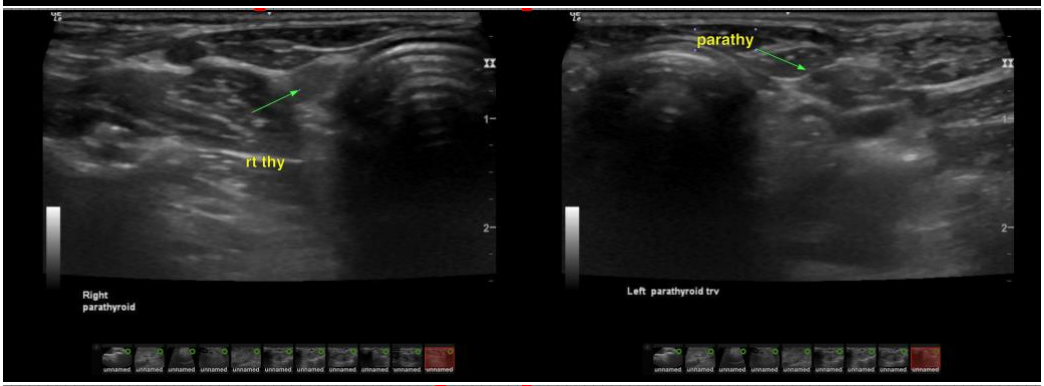
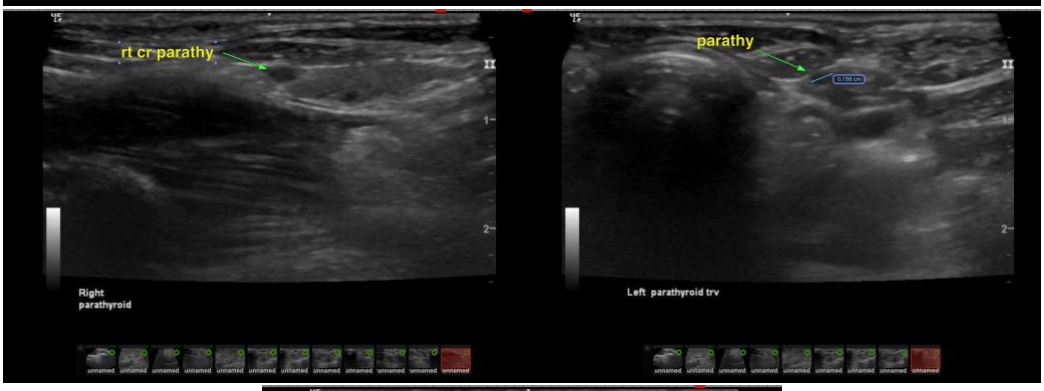
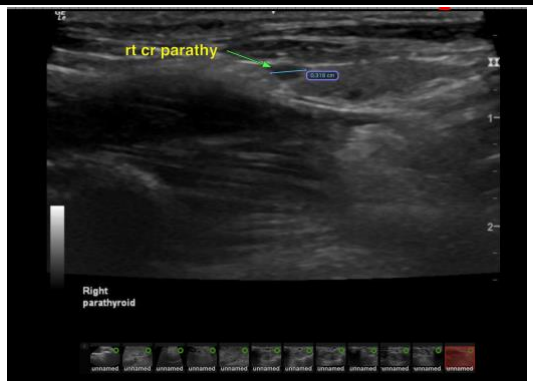
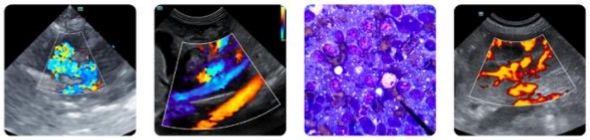








The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not



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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, Owner, Founder -- SonoPath.com
info@SonoPath.com

CANINE HYPERCALCEMIA

http://www.sonopath.com/CanineHypercalcemia

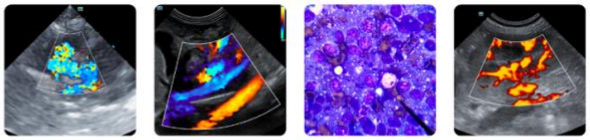
Description: Hypercalcemia is defined as either a persistently elevated total calcium serum (> 12 mg/dl) or ionized calcium (> 1.45 mmol/l) concentration. Clinical signs are often absent with mild hypercalcemia (< 13 mg/dl). In fact, hypercalcemia is often only discovered when serum biochemistry is done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

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



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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

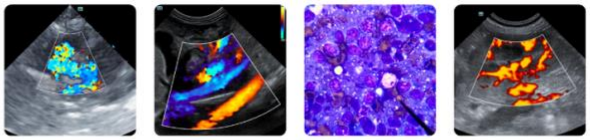
Spurious

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.



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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:

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