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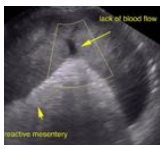

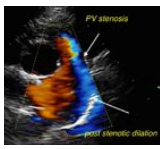

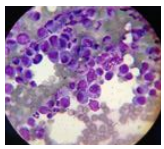
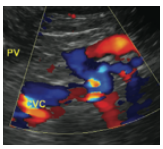

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1-800-838-4268 info@sonopath.com

PATIENT

Kirra Whitehead

SPECIES

Canine

BREED

Mix

SEX

Spayed female

AGE

12 years

WEIGHT

47 lbs

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Dr. Kitz

HOSPITAL NAME

Woodlands AH

REFERRING VET

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INVOICE

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PRESENTING CLINICAL SIGNS

History: lethargic, with lower endurance changes in hair growth with areas of poor regrowth historically elevated liver enzymes historically elevated LDDS - owners elected not to treat at that time panting excessively patient is new to our practice so we rechecked labwork and recommended ultrasound to look at adrenals and liver/GB
Abnormal PE/Chem/CBC/UA Results: patchy areas of thinned hair on dorsal trunk with suspected calcinosis cutis lesions heavy panting and restlessness pot-bellied appearance with thinner skin on abdomen ALKP-3288 (5-131) ALT-818 (12-118) AST-82 (15-66) GGT-15 (1-12) Pre bile acids - 21.2 (0-14.9) Post bile acids -27.2 (0-29.9) BUN-30 (4-27) Creatinine 1.4 (0.5-1.6) T4- 1.6 (0.8-3.5) USG-1.011 trace proteinuria LDDS - resting cortisol -5.1 4 hr post-2.9 8 hr post - 2.3 (no reference ranges provided from lab)

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 was noted. Ureteral papillae were normal.

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 kidneys measured 6.0 cm each.

Adrenal Glands

The left **adrenal gland** was at the upper limits of normal and measured 0.78 cm at the cranial pole and 0.7 cm at the caudal pole. The right adrenal gland was at the upper limits of normal and measured 0.85 cm at the mid body and 1.0 cm at the cranial pole.

Spleen

The **spleen** was normal size and relatively normal contour with multifocal hyperechoic areas of mineralization. This is a benign change; however, can be related to Cushing's disease or other endocrinopathies.

Liver

The **liver** revealed coarse architecture with increased portal markings. Nodular hepatic changes were noted. The gallbladder and common bile duct were unremarkable.

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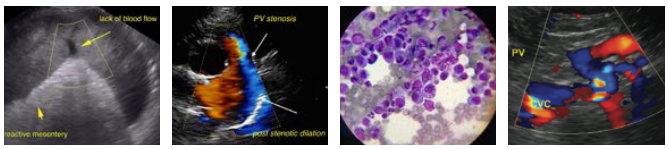

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Gastrointestinal

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.

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.

ULTRASONOGRAPHIC FINDINGS

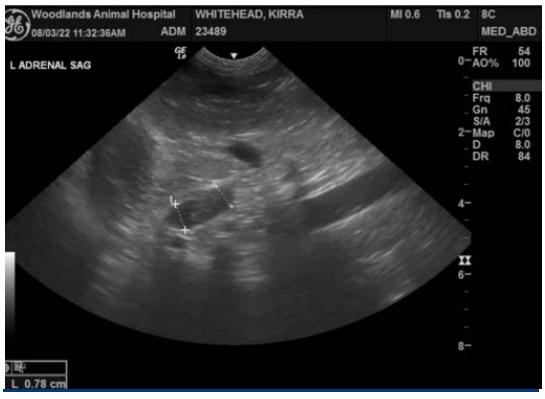
Non-specific, hepatic remodeling.

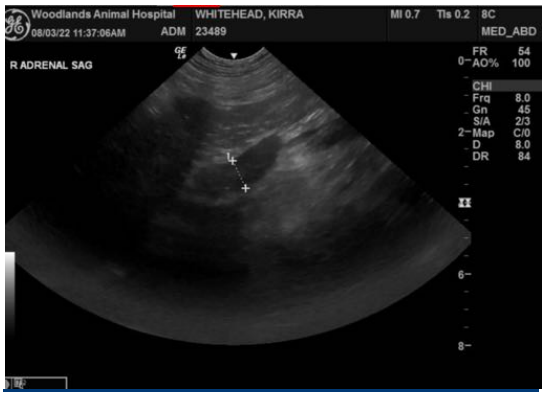
Age related renal changes with mineralization.

Mild bilateral adrenal hypertrophy.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

There is concern for emerging Cushing's/PDH in this patient. FNA of the liver is indicated. Given the bilateral adrenal enlargement the hepatic presentation, splenic presentation, dermal presentation and isosthenuria as well as excessive panting all indicate that this is likely pituitary dependent hyperadrenocorticism. Blood pressure measurements are recommended.





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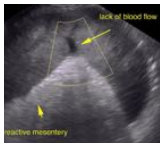
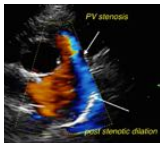
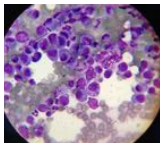
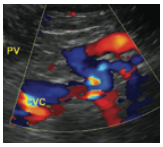

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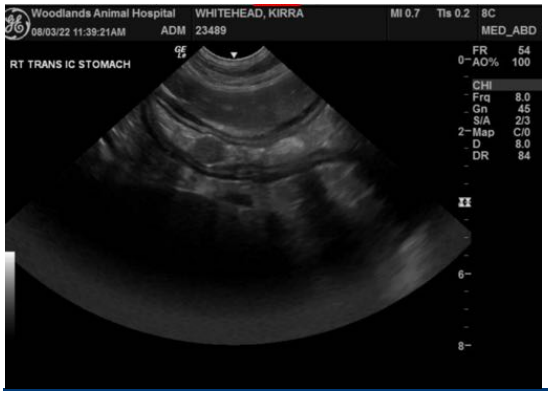

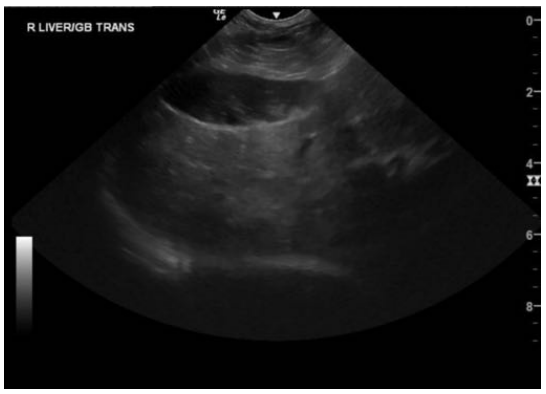

The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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

The following is an applicable excerpt from the *Curbside Guide to Diagnosis & Treatment of Sonographic Disease* offered by SonoPath.com Lindquist, Frank, L and Modler.

An essential quick guide for every general practitioner and sonographer.

https://sonopath.com/products/curbside-guide-editing-due-release-12012015

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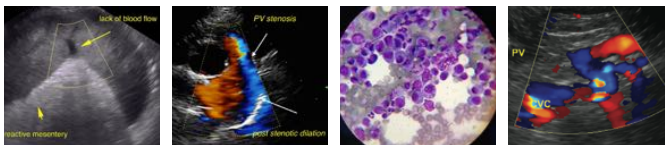

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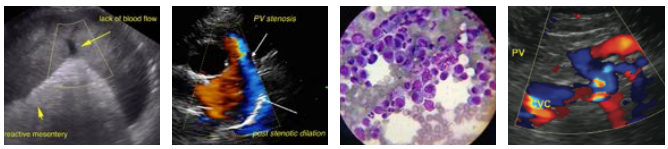
Canine Pituitary-Dependent Hyperadrenocorticism

http://www.sonopath.com/PDH

Long axis of the left adrenal gland in a Yorkshire Terrier
pituitary dependent hyperadrenocorticism and bilateral ad
hyperplasia. Note the symmetrical enlargement of the cra
and caudal pole beyond 8mm in width. No echoarchitectu
changes are noted other than uniform adrenal swelling.

Description: Pituitary-dependent hyperadrenocorticism (PDH) (Cushing's disease) is responsible for 85-90% of all hyperadrenocortoicoid cases in dogs and cats. Typically, a benign functional pituitary tumor results in excessive stimulation of the hypothalamic-pituitary axis, usually leading to bilateral adrenal hypertrophy; however, a macroadenoma can also precipitate bilateral adrenomegaly.

Clinical Signs: PDH most commonly occurs in dogs over 6 years of age and usually at an average age of 9-11 years or older. The most common clinical signs include polyuria and polydipsia (PU/PD) (USG < 1.025 in repeated samples), polyphagia, lethargy, tachypnea, and calcinosis cutis. Physical exam abnormalities typically include hepatomagely, alopecia, a potbellied appearance with fat redistribution, thin skin, pyoderma, and muscle atrophy and weakness. Clinical disease is present when suspicion arises from physical exam findings and clinical signs reported by the owner as well as other possible negative sequelae, such as hypertension, persistent urinary tract infections, proteinuria, and thromboembolic disease. Patients with concurrent diabetes may experience insulin resistance and difficulty controlling the diabetes. In the face of a macroadenoma of the pituitary gland, certain neurological signs may manifest, such as circling, ataxia, behavioral changes, decreased appetite, and wandering.



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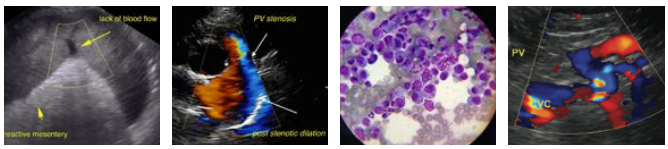
Diagnostics: A minimum database includes a CBC, biochemical profile, urinalysis, and urine culture and sensitivity. Typically, a CBC will reveal the following laboratory abnormalities: a mild elevation in RBC count, neutrophilia, thrombocytosis, lymphopenia, and eosinopenia. Abnormal serum chemistry results include increased ALP, ALT, cholesterol, triglycerides, and glucose in the case of diabetics. The urinalysis will reveal a reduced urine specific gravity, usually below 1.020, and evidence of infection, even in the absence of pyuria. A urine culture should be performed since these patients frequently have occult urinary tract infections. Concomitant proteinuria may be present and an elevated urine protein-creatine (UP:C) ratio may be indicative of PDH. Blood pressure should be measured since hypertension is a common complication of PDH. Since patients are usually older dogs that may have additional pathologies, thoracic radiographs should be performed, especially if they display panting (i.e., tachypnea) as a clinical complaint. In severe cases, thromboembolic disease may be present as well.

Clinical suspicions can be confirmed by adrenal gland function testing as well as abdominal ultrasound. Various tests assess the hypothalamic-pituitary axis. The urine cortisol-creatinine ratio test (UC:Cr) is excellent for ruling out Cushing's disease, especially in patients displaying some abnormal laboratory data but where clinical suspicion is perhaps not high. The UC:Cr has 75-100% sensitivity and 20-25% specificity, which means that false positive results are frequent; however, a negative test rules out hyperadrenocorticism. The finding of two consecutive elevated UC:Cr values improves the sensitivity of the test to 99%. A positive test must be confirmed with additional adrenal gland function testing. The urine sample should be collected at home so that the stress release of cortisol does not result in a false positive.

The low-dose dexamethasone suppression test (LDDST) is the preferred screening test, unless one is concerned about iatrogenic Cushing's disease due to the exogenous administration of a steroid. The sensitivity of the LDDST is reported to range between 85-100% and the specificity between 44-73%. Depending on the study, the sensitivity of the ACTH stimulation test ranges between 57-95% and the specificity between 59-93%. The sensitivity of the ACTH stimulation test in dogs with PDH is higher at 80-83%; it is lower for dogs with adrenal tumors (AT) (i.e., it varies between 57-63%). Thus, as a screening test, an LDDST would be preferred due to its overall better sensitivity. An inverse pattern has been described in a low number of cases in which the 4-hour cortisol level is increased, but the 8-hour sample is below the cutoff value. This is suggestive of PDH and should prompt further testing. The ACTH stimulation test is the only test that discriminates between naturally occurring and iatrogenic hyperadrenocorticism. Unfortunately, since no test has 100% sensitivity and 100% specificity, it may be necessary to complete an extensive workup to confirm the diagnosis. Given the fact that concurrent disease may potentially cause a false positive in all screening tests, then one might consider performing a sonogram first, after verifying the occurrence of true PU/PD (USG < 1.025) and an elevated UC:Cr with a routine urinalysis. A proactive sonogram would help determine if other causes of PU/PD are playing a role, define the adrenal structure as well as that of other effector organs, such as the liver, which exhibits vacuolar hepatopathy patterns, and assess for mucoceles (the latter are often associated with Cushing's disease). Identifying renal, adrenal, and lower urinary structures upon presentation allows for a global assessment of the urinary tract. This is important, as the results will impact whether the course of treatment should be oriented toward the adrenals or focused elsewhere. Given that each medical therapy for Cushing's disease carries some risks, it is imperative that one arrive at an accurate diagnosis.

Ultrasonography for differentiating PDH from AT: Abdominal ultrasound is the most expedient way to differentiate PDH from AT; however, ultrasound alone cannot confirm the functionality of the adrenal gland, which requires specific hormone testing. The LDDST may distinguish PDH from AT in some cases,



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specifically when the 4-hour post-dexamethasone cortisol concentration is below the laboratory cutoff or less than 50% of the baseline cortisol measurement, or if the 8-hour cortisol measurement is less than 50% of the baseline cortisol measurement, but greater than the laboratory cutoff. Suppression of cortisol on the high-dose dexamethasone suppression test (HDDST) is considered when the 4- or 8-hour cortisol measurement is below the laboratory cutoff or less than 50% of the baseline cortisol level. An endogenous ACTH level is another discriminatory test; however, the assays will vary in technique and hence sensitivity of detection varies. In addition, the molecule is labile and requires special handling, so it is crucial to follow the laboratory's recommendations regarding sample collection.

On ultrasound examination, dogs with PDH typically show symmetrically enlarged adrenal glands. Adrenal glands are considered to be enlarged when their width exceeds 0.8 cm in large breeds and 0.6 cm in small breeds. (These size parameters are a solid rule of thumb; however, in a minor number of cases, or if it is early on in the disease, dogs will present with a normal adrenal size.) Moreover, excessive adrenal size does not necessarily mean the patient has Cushing's disease. Hyperplasia, adenoma, stress, and age-related remodeling can all cause alteration of the adrenal structure. Adrenocorticoid tumors comprise the remainder of Cushing's cases and tend to present as unilateral adrenal hypertrophy with contralateral atrophy; however, bilateral AT can occur in 10-15% of cases.

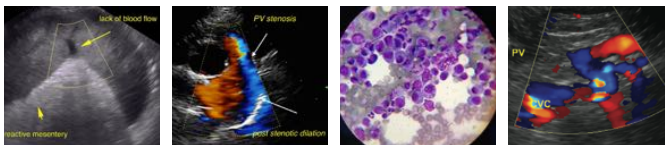
Dogs with chronic illness may also have symmetrically prominent adrenal glands. Thus, prominent adrenal glands themselves should not constitute the sole diagnostic test for PDH. Approximately 72% of adrenal tumors may be identified with abdominal ultrasound. Results of the ultrasound may be confounded in patients that have bilateral adrenal tumors, concurrent PDH and AT, macronodular hyperplasia of the adrenal glands, or ectopic ACTH secretion. Other common ultrasound findings in dogs with PDH include hepatomegaly, dystrophic mineralization, and a distended urinary bladder. Ultrasound-guided FNA of an AT can be performed by experienced clinical sonographers and may help differentiate hyperplasia from a tumor; however, significant side effects, such as an adrenaline surge and hemorrhage, can occur.

Treatment: Treatment is targeted at ameliorating clinical signs and preventing secondary complications, such as hypertension, UTI, skin infection, thromboembolic disease, gall bladder mucocele, increased risk for pancreatitis due to hyperlipidemia, increased risk for diabetes mellitus, increased risk for calcium-containing uroliths, and protein-losing nephropathy.

Frequent follow-ups and laboratory testing are necessary, particularly in the early stages of treatment. Medical therapy is the mainstay of treatment and entails standard doses of either mitotane or trilostane. Other less common forms of treatment include intentional, high-dose treatment with mitotane to non-selectively destroy all three adrenocortical layers, followed by hormonal supplementation, bilateral adrenalectomy, and transsphenoidal hypophysectomy. (The latter, however, is not widely available.)

Medical therapy has traditionally been implemented with mitotane (Lysodren) at 25-50 mg/kg for 7-10 days, then 25 mg/kg given twice a week with dose adjustments primarily based on clinical resolution and ACTH stimulation. The main negative side effects of the medication itself are adverse gastrointestinal effects, such as vomiting and anorexia, or signs of iatrogenic hypoadrenocorticism, which can occur due to adrenal necrosis, thrombosis, and spontaneous hemorrhage.

Trilostane (Vetoryl) was first used to treat PDH in 1998. This medication was originally available in Europe and the UK, with off-label usage in the United States. In December 2008, the FDA approved Trilostane (Dechra Veterinary Products) for use in dogs with hyperadrenocorticism. It is indicated for use in PDH, but also received designation status as a Minor Use Drug for adrenal tumors. Trilostane reversibly and competitively inhibits 3B-hydroxysteroid dehydrogenase, an enzyme that causes a decrease in the



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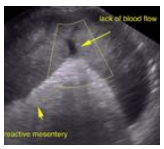
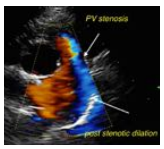
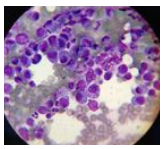
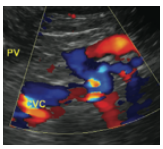
conversion of progesterone to cortisol, aldosterone, and androstenodione; this results in decreased mineralocorticoids and glucocorticoids. Trilostane may influence other hormones along the cholesterol to corticosteroid pathway, as some hormone levels increase with trilostane. In some instances, adrenal size increases with trilostane use due to a lack of negative inhibition from reduced cortisol levels. This will result in a bilateral mass-type presentation on sonographic evaluation.

Because it has fewer side effects, trilostane was initially presented as an alternative to Lysodren. Adverse effects are usually mild and self-limiting, and consist of diarrhea, vomiting, and lethargy. Yet, iatrogenic hypoadrenocorticism has been reported secondary to trilostane use with clinical and biochemical evidence of adrenal necrosis. This has been shown to be reversible in some patients, but permanent in others. In one study, approximately 25% of the dogs treated with trilostane experienced at least one episode of hypoadrenocorticism during the treatment period. Treatment with trilostane was discontinued in 11% of dogs due to the effects of cortisol suppression; however, only 2% of patients required long-term glucocorticoid and mineralocorticoid supplementation. Drug-induced hypoadrenocorticism occurs more frequently in dogs that were treated with trilostane for more than a year. Death secondary to adrenocortical necrosis has been observed; thus, frequent monitoring is critical and precautions should be taken. Deaths of this kind are thought to have been the result of a too aggressive initial dosing scheme. Consequently, subsequent dosing recommendations have been altered to reflect a lower dosage regimen in which the initial dose can be as low as 1-2 mg/kg PO BID. (Note: Since the dosage recommendations and management for this medication continue to evolve, one should consult the most recent references regarding dosages and monitoring.)

In most dogs with PDH, the functional pituitary tumor remains small in size (<1 cm). A macroadenoma is a pituitary tumor that is easily visualized on brain imaging (CT scan) and is greater than 10 mm in size. Although the use of advanced imaging and MRI may enhance our ability to detect smaller lesions, it should be noted that the size of the mass does not always correlate with the advent of clinical signs. In fact, smaller masses sometimes cause neurological abnormalities whereas larger ones do not always result in signs. Yet, the height of the mass is often related to the development of signs; in some cases, a mass with a height of 7-8 mm can cause signs. Neurological signs are reported in 15-20% of dogs with PDH. The development of signs is more likely related to the rate of tumor growth, the size of the skull, and the presence of concurrent edema or inflammation.

The most common clinical signs include dull behavior, listlessness, inappetence or anorexia, apparent disorientation, aimless wandering, staring, and pacing. Ataxia, head pressing, circling, urinating and/or defecating in the home, and grand mal seizures may occur, but are much less common. In rare instances, patients can develop diabetes insipidus, specific cranial nerve deficits, aggressive behavior, thermoregulatory disturbances, or blindness. In some cases, the neurological abnormalities are present at the time of diagnosis; for others, they develop during the course of therapy. As such, one may wish to consider brain imaging for patients with PDH, as earlier intervention with radiation therapy (i.e., with Cobalt-60 Teletherapy or a linear accelerator) generally yields a better clinical outcome. Dogs with more severe signs as well as larger lesions (i.e., 20-25 mm) have a worse prognosis than those with less severe signs and smaller lesions (i.e., less than 20 mm). Given the success of radiation in reducing neurological signs, this modality may be recommended for any dog with a pituitary tumor smaller than 7 mm in size. Prognosis is excellent for dogs without clinical signs and those with masses smaller than 15 mm in size. Concurrent medical therapy is typically still necessary, as radiation therapy alone does not always effectively control the secretion of hormones from the pituitary tumor.

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32160

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Conclusion: Canine hyperadrenocorticism is a common condition in dogs. Diagnosis can sometimes be challenging, but one can typically use a combination of clinical signs, function testing, and imaging to arrive at a diagnosis. Therapy is either medical or surgical. Radiation therapy is advised for patients with pituitary tumors greater than 7 mm.

Long axis of the right adrenal gland of the Yorkshire Terr in the title image. Note the uniform adrenal enlargement beyond 8mm in width. No echoarchitectural changes are noted and the thin, echogenic corticomedullary junction is distinctly visible and intact (arrow).

Long axis of a uniformly enlarged left adrenal gland in a cat with pituitary dependent hyperadrenocorticism. Note t shortened appearance and minor prominence of the crania and caudal pole as compared with the dog. The color Doppler signal shows the blue renal vein entering into the caudal vena cava (arrow), the celiac and cranial mesenteri arteries in the mid field, and phrenicoabdominal artery in t near field.

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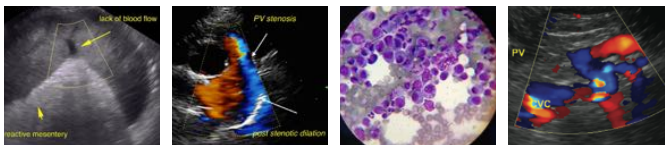

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PATIENT

Kirra Whitehead

SPECIES

Canine

BREED

Mix

SEX

Spayed female

AGE

12 years

WEIGHT

47 lbs

INTERPRETED BY

Eric Lindquist, DMV
DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Dr. Kitz

HOSPITAL NAME

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Long axis of the right adrenal gland in a cat with pituitar dependent hyperadrenocorticism and bilateral adrenal hyperplasia. Mild generalized echogenicity increase and uniform adrenal gland enlargement is noted.

Long axis view of an enlarged left adrenal gland in a geriatric Beagle dog with pituitary dependent hyperadrenocorticism (PDH). The left adrenal width is mildly excessive (normal width 0.75 cm for this size dog) and heterogeneous echogenic nodules are noted within the parenchyma. Yet, no capsular expansion is noted associate with the nodules. This parenchymal remodeling pattern is common finding in PDH adrenals. The cortico-medullary rim cannot be distinguished owing to the remodeling.

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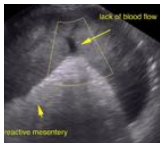
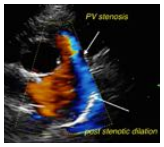
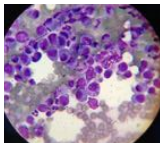
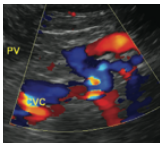

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