Report Requested By

Rocky Road Animal Clinic
Dr's Name: Samantha Smith(Clinic)
Submitted By: Samantha Smith
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Phone: 7037316754
Fax: 123456789
1934 Old Gallows Rd,
Vienna, 22182
Patient: Roscoe Adams
Canine | Schnauzer | Neutered Male | 32 Lbs

Report Provided By

Tyler Anderson, DVM
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PO Box 40398,
Arlington, 22204
Submitted: Oct 21, 2019 15:29 PM
Appointment Date: Oct 21, 19
Closed: Oct 21, 2019 15:32 PM
Case ID: MiVU65-2019-001814

Services

Abdominal Ultrasound
Interpretation provided by MiVU doctors

Clinical Findings

Scheduling Notes (Please Call with Special Requests)
30 minute call ahead

Pertinent Medical History (Laboratory Findings, Radiographic Findings, Current Medications (include dosages)
PU/PD, polyphagia, ALKP 1300, R/O Cushing's disease vs. primary liver

Will this patient require sedation?
No

If Yes, Have you obtained owner approval for sedation?
No

If fine-needle biopsies are recommended, have you obtained owner approval to collect samples for cytology?
Yes

Has the patient been fasted for >12 hours?
Yes

Exam

Liver

Moderately increased size, mildly rounded shape and moderately coarse hyperechoic echogenicity. No focal lesions are appreciated. The gall bladder is clean and of normal size and shape.

Kidneys

Normal size and shape with normal corticomedullary dimensions. No pyelectasia visualized.

Spleen

Normal size, shape, and echogenicity. No focal lesions appreciated.

Urinary Bladder

The bladder is of relatively normal contour and thickness. No overt obstruction, uroliths, or neoplasia noted.

Adrenal Glands

Both adrenal glands were visualized and recognized as having abnormally rounded "plump" shape, increased size (Lt/Rt = 7.8/7.9mm), normal position with stimulated overall echogenicity for this breed. No adrenal invasion into the vena cava, phrenic vein thrombosis, dystrophic mineralization or clinically significant nodular changes were noted.

Pancreas

No significant findings. Isoechoic to peripancreatic fat.

Intestinal Tract

The stomach, small intestinal loops and colon are WNL - normal bowel layering, thickness, and motility.

Serosal Surfaces

WNL

Prostate

Normal size, shape, and echogenicity.

Report

Abdominal Ultrasound Interpretation

Liver - the findings are moderate - DDx:

a) Chronic vs. Acute hepatitis or cholangiohepatitis (bacterial vs. sterile vs. toxin)
b) Steroid hepatopathy / Vacuolar hepatopathy / Glycogen storage disease / Copper storage disease
c) Diabetes mellitus
d) Infiltrative neoplasia (lymphosarcoma)
e) Fungal infection

Adrenals DDx: bilaterally enlarged and stimulated adrenal glands are suggestive of pituitary-dependent hyperadrenocorticism. If the patient doesn't have clinical signs for hyperadrenocorticism, stress may be the etiology for the adrenomegaly.

Recommendations

Consider running an ACTH or LDDS test to confirm hyperadrenocorticism before initiating therapy. Pituitary dependent hyperadrenocorticism is suspected in this case.

Consider other diagnostics/therapeutics as clinical signs dictate.

Articles/Comments

Diagnosing Canine Cushing's Syndrome - Timothy A. Allen, DVM, DACVIM (SAIM), Dechra Veterinary Products

Hyperadrenocorticism (HAC) is the term used to describe the clinical and laboratory changes associated with chronic exposure to glucocorticoids. A synonym for HAC is Cushing's syndrome. Hyperadrenocorticism can be either sPontaneous (naturally occurring) or iatrogenic. There are two forms of spontaneous HAC. Pituitary dependent (PDH) is the most common form (80-85% of cases) and is due to hyperplasia of the adrenal cortex resulting from inappropriate release of adrenocorticotropic hormone (ACTH) by a pituitary tumor (usually benign). The other form of HAC is adrenal dependent (ADH) (15-20% of cases), which is due to a functional tumor of the adrenal cortex. The tumor secretes cortisol independent of the normal pituitary control (feedback) process. Adrenal tumors can be either benign or malignant. Iatrogenic HAC results from chronic administration of glucocorticoids (oral, parenteral or topical, including otic and ophthalmic).

Clinical Signs

Typical clinical findings include polydipsia, polyuria, polyphagia, excessive panting, pot-bellied appearance, bilaterally symmetrical alopecia (hair loss/thin hair coat-typically, non-pruritic and predominantly truncal), thinning of the skin, muscle wasting, weakness, and lethargy. Less frequent clinical signs include comedones, calcinosis cutis and a plantigrade stance. Common complications of HAC include pyoderma, urinary tract infection, proteinuria, hypertension, diabetes mellitus and thromboembolism.

The CBC reveals a "stress leukogram" (mature neutrophilia, monocytosis, lymphopenia and eosinpenia), mild polycythemia and thrombocytosis. Typical changes in the serum biochemistry panel include increased alkaline phosphatase activity, increased alanine transaminase activity (usually less than twice the upper limit of the reference range), increased triglyceride concentration, increased cholesterol concentration and slightiy decreased urea nitrogen concentration.

Diagnosis

Hyperadrenocorticism is diagnosed based on the Presence of clinical signs, suggestive findings on CBC and serum biochemistry profiles and a positive confirmatory screening test (ACTH stimulation test, low dose dexamethasone suppression test-LDDST or urinary cortisol:creatinine test-UCCR).

There is no perfect screening test for the diagnosis of HAC. Many veterinary endocrinologists recommend the LDDST as the initial test, however, the choice of which screening test to use is influenced by several considerations including the following:

1. Number and severity of clinical signs suggesting HAC
2. Ongoing or recent treatment with glucocorticoids
3. Strength of suspicion of an adrenal gland tumor
4. Potential presence of concurrent non-adrenal illness

Results of the ACTH stimulation test will be in the normal range (false negative) in roughly 20% of dogs with HAC. The false negative rate for the LDDST is lower; however, the specificity of the LDDST is poor (between 44% and 73%).ln general, if a patient has serious nonadrenal disease, the more likely the LDDST will be falsely positive. The urinary cortisol to creatinine ratio (UCCR) is best used to rule out HAC. Nearly 100% of dogs with HAC have an increased (positive) UCCR. Unfortunately, many dogs with an increased UCCR do not have HAC. If the results of one of the specialized endocrine tests are normal, but clinical suspicion of HAC is high, consider performing the other screening endocrine test.

TEST SENSITIVITY* (%) / SPECIFICITY^ (%)

ACTH Stim - Less than ideal (80-95) / Good (86-91)

LDDST - Excellent (85-100) / Poor (44-73)

UCCR - Excellent (75-100) / Poor (24-77)

*Sensitivity > percent of positive tests in dogs with HAC

^Specificity > percent of negative tests in dogs without HAC

ACTH Stimulation Test-The forms of corticotropin (ACTH) historically used for testing are corticotrophin gel (Actharo) or synthetic corticotrophin (cosyntropin). Many veterinary endocrinologists use Cortrosyn (cosyntropin manufactured by Amphastar Pharmaceuticals, Rancho Cucamonga, CA). Cortrosyn is sold in packs of l0 or as a single vial of 0.25 mg (250 ug). Cosyntropin can be reconstituted and stored frozen (-20"C) in plastic syringes for up to 6 months. The dose for dogs is 5 pg/kg IV or IM (maximum dose per dog 250 pg). A pre-ACTH sample is collected, ACTH is injected IV and one post-ACTH sample is collected one hour later.

Four compounded ACTH formulations sold to veterinarians have been tested in normal dogs. These four formulations consistently stimulated increased cortisol concentrations at one hour, but yielded variable results at two hours.

LDDST-Either dexamethasone sodium phosphate or dexamethasone in polyethylene glycol can be used, but the dose is based on the amount of active dexamethasone. For example , a 4 mglmL solution of dexamethasone sodium phosphate contains about 3 mg/ml of active dexamethasone. In order to give the correct dose in small dogs, make a 1:20 dilution by mixing 0.2 mL of dexamethasone with 3.8 mL of sterile water or saline. For example, for Azium@ a 1:20 dilution will make the final solution 0.1 mg/ml dexamethasone. In order to make a diagnosis of HAC, the dog must have clinical signs consistent with the disorder. If a dog has a positive screening test but doesn't have clinical signs of HAC, the dog either does not have HAC or it may have subclinical HAC. In the latter case, monitoring for the development of clinical signs and repeat endocrine testing are recommended.

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