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**SPECIAL
POINTS OF
INTEREST:**

- **HAC** is one of the most commonly diagnosed endocrine problems in canine veterinary patients
- Clinical signs for classic and atypical Cushing's are the same.
- The diagnosis of atypical Cushing's includes a normal ACTH stimulation and LDDS test in addition to marked increases in at least 2 of the adrenal sex hormones

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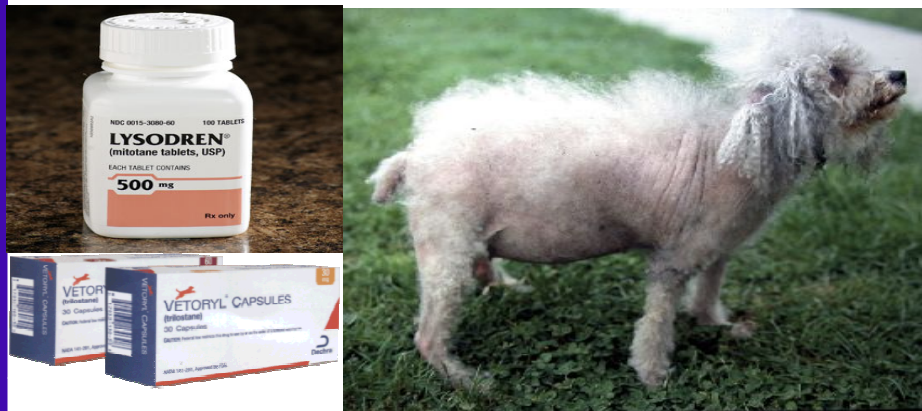
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Canine Atypical Cushing's



Dear Colleague,
Hyperadrenocorticism (HAC) is a common canine endocrine condition—however it is not always easily diagnosed. Recent advances in our understanding of the steroid production pathway have made us realize that not all Cushing's patients will have a typical or expected ACTH or LDDS test result. In fact, further testing which allows us to evaluate for sex steroid abnormalities, is causing us to reclassify some patients as having "atypical" Cushing's disease.

What is classic hyperadrenocorticism (Cushings)?

Classic hyperadrenocorticism (HAC) or Cushing's disease, is one of the most common endocrinopathies of dogs. It is characterized by excessive cortisol secretion from the adrenal gland(s). This typically occurs as a result of a benign pituitary tumor, however less commonly an adrenal tumor or malignant pituitary tumor can be responsible for the excess cortisol. Although any breed can be affected, this disease is seen with increased frequency in smaller breed dogs (Poodles, Dachshunds and Boston Terriers). Clinical signs of HAC (both the classic and atypical forms) include polyuria/polydypsia, polyphagia muscle weakness and dermatological changes. Dogs with "classic" Cushing's disease can typically be diagnosed with routine screening tests which include an ACTH stimulation test or a low dose dexamethasone suppression test. A urine creatinine cortisol ratio can be used to know if more advanced endocrinology testing is needed—but cannot be used exclusively to diagnose HAC.

Atypical Cushing's Disease

The clinical signs associated with increased concentrations of adrenal sex steroids are indistinguishable from those of glucocorticoid excess. Although the pathophysiology behind atypical HAC is not well understood, there are two proposed theories. The first theory is that progestagens have intrinsic glucocorticoid activity. As a result, an excess amount of progestagens will mimic cortisol excess and as a result, these patients will exhibit signs of HAC. The second theory is that progestagens increase the availability of cortisol by binding to cortisol receptors or displacing cortisol from its binding proteins.

Atypical Cushing's disease should be considered as a diagnosis in dogs that exhibit clinical signs of HAC along with laboratory values that support HAC however cortisol excess cannot be demonstrated by an ACTH stimulation or LDDS testing.

Testing for Atypical Cushing's Disease

When historical, physical examination and routine laboratory findings are suggestive of HAC, routine pituitary-adrenal endocrine function testing (ACTH stimulation or LDDS) is indicated. In many dogs, in the absence of concurrent illness, the diagnosis of HAC is straight-forward. In dogs with clinical signs suggestive of HAC that have normal ACTH stimulation and low-dose dexamethasone suppression test results, measurement of serum concentrations or other adrenal steroid hormones may assist in diagnosis.

Most dogs with classic HAC have *mild* elevations of two or three adrenal steroid hormones in addition to cortisol, both before and after stimulation with ACTH. Dogs with non-adrenal illness may also have increased values but typically only for 17-hydroxyprogesterone secretion. In dogs suspected of having atypical HAC, marked increases (1.5 to 2 times the high end of the reference interval) in two or three adrenal steroid hormones is supportive of a diagnosis of atypical Cushing's disease. All elevations must be considered in light of a dog's gender and neuter status, as well as the presence or absence of any non-adrenal illness and appropriate reference intervals applied.

The most extensive adrenal steroid hormone profile is offered by the Clinical Endocrinology Service at the University of Tennessee. The protocol for running the profile is the same as for a standard ACTH stimulation test but requires a larger sample (2 mL of serum).

Treatment for Atypical Cushing's Disease

Dogs with pituitary-dependent atypical HAC respond well to routine therapy for classic HAC. Good clinical responses have been reported with both mitotane and trilostane and no advantage of one over the other has been documented. Treatment efficacy is monitored with standard ACTH stimulation testing. Repeat adrenal steroid hormone profiles are not recommended since values do not tend to correlate with treatment efficacy. The goal of treatment is improvement or resolution of clinical signs and suppression of cortisol concentration below the reference range for post-ACTH cortisol (<2-6 mcg/dL). In dogs with adrenocortical tumors, whether typical or atypical, the treatment of choice is surgical resection. Although some cortisol-secreting adrenal tumors are amenable to medical therapy, noncortisol-secreting adrenal tumors tend to respond poorly.

How can I refer a patient for further evaluation?

If you have any questions concerning Cushing's disease (classic and atypical) or would like to refer a patient for continued evaluation and testing, please call our Internal Medicine team at 713-693-1144.

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