

Introduction to Canine Hyperadrenocorticism (*Cushing's Syndrome*)

Cause

Hyperadrenocorticism results from overproduction of adrenal glucocorticoids. Approximately 80% cases are caused by an ACTH secreting pituitary tumour. This excess of ACTH causes bilateral symmetrical adrenal enlargement and hyper-secretion of cortisol. The remaining cases are due to a primary adrenal tumour.

Signalment and Clinical Signs

Clinical signs can be extensive but include polyuria, polydipsia, polyphagia, generalised alopecia, hepatomegaly, muscle weakness, pot belly, thin skin, bruising and in some cases neurological signs consequent upon a pituitary tumour.

Diagnostic Tests

It is crucial to understand that all of the diagnostic tests have limitations and all can give both false negative and false positive results. It is therefore particularly important to use the tests to support a clinical suspicion of hyperadrenocorticism:

The diagnosis should not rely upon the results of blood tests alone.

Routine biochemistry and haematological abnormalities

Increases in ALKP present in 80-90 % cases. Mild hypercholesterolaemia also common. Typical stress leukogram (*mature neutrophilia, lymphopenia and eosinopenia*) often seen.

ACTH stimulation test

Collect fasting whole blood sample, administer 250µg synthetic ACTH intramuscularly, collect a second blood sample one hour later. Request cortisol assay on pre and post samples.

Low dose dexamethasone suppression test

Collect fasting whole blood sample, administer 0.015 mg/kg dexamethasone intravenously, collect additional blood samples three and eight hours later. Measure cortisol in each sample.

The interpretation of the ACTH stimulation and low dose dex suppression tests is influenced by the history, routine laboratory findings, and recent drug therapy. Please provide Axioms clinical pathologists with the relevant history to help get the most out of the tests performed.

Treatment

Surgical removal of an adrenal tumour can be considered in those patients with no evidence of metastatic spread. This is an intensive procedure requiring appropriate facilities but is potentially curative. Most dogs are medically managed. Vetoryl (*Trilostane*) has recently been licensed (*see Axiom Endocrine Factsheet 2.1*). Mitotane (*Lysodren*) therapy remains commonly used drug for treating HAC. Starting dose is approximately 50mg/kg daily for 7-10 days. Once initial control is achieved this dose can be decreased to weekly in most dogs.

Therapeutic Monitoring

Monitoring of therapy should consist of clinical evaluation and results of an ACTH stimulation test. In dogs receiving Trilostane treatment, the ACTH stimulation test should be performed 4-6 hours after giving that day's medication. Cortisol results from 50-100 nmol/L pre and post-ACTH are indicative of good control if receiving mitotane therapy.