

Addison's Disease

What is addison's disease?

Addison's disease (also called primary hypoadrenocorticism) is a condition caused by the deficiency of hormones made by the adrenal gland. The adrenal glands (there are two, one in front of each kidney) makes several important hormones. With Addison's disease, clinical signs occur due to the inability of the adrenal gland to make adequate amounts of the hormones cortisol and aldosterone. These two hormones are critical to maintaining normal body functions. Cortisol helps to combat stress, ameliorate inflammation, and make available energy stores to the body by encouraging the breakdown of muscle and fat. Aldosterone helps the body reabsorb sodium (a very important electrolyte) and water (thus maintaining normal fluid balance). Aldosterone also helps the body excrete excess potassium (another important electrolyte). With a deficiency in aldosterone the body becomes sodium and water deficient (life threatening dehydration may occur) and hyperkalemia develops (elevated potassium levels in the blood). Hyperkalemia may cause slowing of the heart rate and the development of cardiac arrhythmias.

What causes addison's disease?

Addison's disease (also called primary hypoadrenocorticism) results from destruction of the outermost layers of the adrenal gland. The two most common causes of destruction include immune-mediated disease (where the body launches a "self-attack" against the adrenal gland for unknown reasons) or drug therapy. The drugs most likely to cause Addison's disease are Lysodren and Trilostane (these drugs are used to Cushing's disease which is associated with excessive cortisol production). Other causes are possible as well, but less common. Lastly, there is another form of this disease called secondary hypoadrenocorticism. In this form of the disease, the problem stems from an ACTH deficiency. ACTH is a hormone made by the pituitary gland and acts to stimulate the adrenal gland to make cortisol. Without adequate amounts of ACTH, the adrenal gland is not stimulated to make cortisol. Clinical signs of this form of the disease are more mild (as there is only a cortisol deficiency, not an aldosterone deficiency).

What are common clinical signs?

The clinical signs of Addison's disease are quite variable depending on the stage (or severity) of disease. Patients that are more mildly affected may exhibit intermittent exercise intolerance, poor appetite, increased thirst and urination (associated with increased sodium and water loss in the urine), vomiting, and diarrhea. As the disease advances and electrolyte (low sodium, high potassium) abnormalities become more profound, the patient may become severely dehydrated, suffer hypovolemic collapse, have a low heart rate, and develop cardiac arrhythmias. Each patient is a bit different in the symptoms that they show, and how quickly the clinical signs progress. Some patients may appear to be in renal failure as the severe dehydration due to Addison's disease causes elevation of blood products (BUN and creatinine) that are frequently noted with renal failure. The clinical signs of secondary hypoadrenocorticism are usually more mild and vague in nature, but may include intermittent lethargy, depression, weakness, nausea and sometimes vomiting and diarrhea.

How is addison's disease diagnosed?

The diagnosis of Addison's disease usually involves several steps (as this disease may mimic several others). Initial screening tests (CBC, biochemistry profile, urinalysis) along with clinical signs offer the first clues that this disease may be present. Common findings noted on initial lab work include electrolyte abnormalities (low sodium, high potassium), variably low glucose, and variable elevations in BUN and creatinine (depending on dehydration status). With the secondary form of disease, the lab abnormalities present are usually more

subtle. Specialized testing (an ACTH stimulation test) is required to establish a definitive diagnosis. With this test, a resting cortisol value is obtained, ACTH is given, and then a post-cortisol value is obtained. In a normal animal, there will be a “surge” of cortisol released into the blood (if the adrenal gland is working okay) in response to the ACTH. If the adrenal gland is not working properly, there will be a notable absence of the post-cortisol surge. This absence confirms the diagnosis of Addison’s disease.

How is addison’s disease treated?

The treatment of choice for the patient with Addison’s disease is to supplement with both a glucocorticoid (cortisol-like drug) and also a mineralocorticoid (aldosterone-like drug). There are two options to supply these hormones. The first is to administer an injectable mineralocorticoid (DOCP – Percorten-V). Injections are carried out lifelong and usually at a schedule of about every 25 days. As these injections do not contain glucocorticoids, it is also important to send home oral glucocorticoids lifelong (such as prednisone or prednisolone). The other approach is to use an oral mineralocorticoid (Fludrocortisone - Florinef) for longterm treatment. Florinef also has some glucocorticoid activity (so some patients do not require additional oral prednisone or prednisolone). Treatment with either option (particularly in larger dogs) can be somewhat expensive due to drug costs. Lastly, no matter what treatment route is selected, “extra” doses of prednisone or prednisolone need to be made available to the patient in times of “increased stress” (emotional or physical stresses) as the diseased adrenal gland can no longer respond to stress with a protective “surge” release of cortisol. Treatment of secondary hypoadrenocorticism is simpler and just involves administration of oral glucocorticoids (although as for Addison’s disease, extra amounts should be available during episodes of stress).

Recheck evaluations (physical exam, electrolyte levels) on a consistent basis are necessary to adequately manage this disease. In general, for the oral form of medication (Florinef) recheck electrolytes are monitored every one to two weeks until the patient is stable. In the patient on the injectable form of medication (DOCP), electrolytes are checked midway through therapy and then again on the day of the next scheduled injection until stable. Once the Addisonian patient is stable on medications, recheck electrolytes can be checked every 4 months or so thereafter. The patient should also be monitored at home for signs of poor control (depression, poor appetite, listlessness, decreased exercise tolerance, increased drinking or urination, etc.). If signs of poor control are evident, a recheck visit with the veterinarian should be scheduled immediately.

What is the prognosis for dogs diagnosed with addison’s disease?

The overall prognosis for the patient with Addison’s disease is quite good when consistent maintenance therapy and recheck evaluations are carried out.