Addison's Disease (Hypoadrenocorticism)

The two terms will be used interchangeably through this text

Adrenal Hormones

The adrenal gland is so named because it is located just forward of the kidney (renal means kidney). The center of the gland is called the medulla, and the outer area is called the cortex. While both areas produce hormones, Addison's disease concerns the hormones produced by the cortex; these hormones are called corticosteroids and mineralocorticoids.

Corticosteroids are the hormones that enable us to adapt physiologically to stress. The glucocorticoids (such as cortisol) gear the metabolism towards preparing to burn rather than store fuels so as to be ready for a "fight or flight" situation. Corticosteroid hormones are needed to adapt to stressful situations and without these hormones, even small stresses could lead to physiologic disaster.

The mineralocorticoids influence the electrolytes sodium and potassium. They also regulate water homeostasis (maintaining a normal water balance).

Hypoadrenocorticism (Addison's Disease)

In animals with Addison's disease, there is a deficiency of the corticosteroid hormones. It is unusual to discover the direct cause of this deficiency unless the patient is taking medications that disrupt adrenal balance (like ketoconazole, Lysodren or trilostane).

Clinical Signs

At first signs are vague and can include the following symptoms: poor appetite, lethargy, weight loss, weakness, vomiting, diarrhea, collapse, and regurgitation. Ultimately, the disease can result in a phenomenon known as the Addisonian crisis. The animal collapses in shock due to its inability to adapt to the caloric and circulatory requirements during stress. Blood sugar may drop dangerously low. Potassium levels soar and disrupt the heart rhythm. The patient may not survive this episode. In 25% of dogs, the symptoms can wax and wane, making the diagnosis more difficult.

Making the Diagnosis

Veterinarians are typically presented with a young animal in shock. There is usually no history of trauma or toxic exposure, so general treatment for shock is initiated. This consists of rapid administration of fluids (usually lactated ringers solution, which has little potassium and a moderate amount of sodium) plus some glucocorticoids. By coincidence, treatment for shock happens to be similar to the specific treatment for Addison's disease, so often the patient recovers without the veterinarian really knowing why.

The blood panel can come back showing elevations in the renal parameters (BUN and creatinine) and elevated potassium, which is suggestive of acute renal failure. The veterinarian may become suspicious of another diagnosis as the patient will respond well to fluid administration where as most renal failure patients do not respond as well and have a poor prognosis.

The only definitive test for Addison's disease is the ACTH stimulation test. The patient receives a dose of ACTH, the pituitary hormone responsible for the release of corticosteroids in times of stress. A normal animal will show an elevation in cortisol in response to ACTH while an Addisonian has no corticosteroids to respond with. This lack of response is diagnostic for Addison's disease.

Treatment

Treatment for hypoadrenocorticism is the replacement of the missing mineralocorticoids and glucocorticoid hormones. The most common way to treat this condition is with an injectable medication called DOCP (brand name Percorten-V). This treatment is given approximately every 25 days. Electrolytes are measured every few weeks at first, but testing can usually eventually be tapered to once or twice a year. Most dogs also require glucocorticoid supplementation (such as a low dose of prednisone). Treatment with both medications is life-long.

What is Atypical Addison's Disease?

Approximately one dog in 42 will have a certain form of Addison's disease called "atypical".

In this form of the disease the patient is only deficient in the glucocorticoids. Diagnosis is still by ACTH stimulation test and an endogenous ACTH level. Treatment is supplementation of glucocorticoid hormones, such as prednisone. It should be noted that often these patients ultimately progress to the more typical Addison's disease with electrolyte imbalance.

A similar deficiency in glucocorticoids (but not mineralocorticoids) results when a pet has been on long-term oral glucocorticoids (such as prednisone) and medication is discontinued too abruptly. Long-term glucocorticoid use leaves the outer layers of the adrenal cortex inactive and unstimulated by the pituitary gland, since pills or shots are providing the body with more than enough glucocorticoids. Once the medication is withdrawn, the body is back to relying on its own adrenal glands for glucocorticoids but the gland has atrophied from lack of stimulation. This creates a deficiency in glucocorticoids similar to Atypical Addison's disease and is the reason why steroid hormones are typically tapered off rather than abruptly discontinued.