Addison's: The "Other" Adrenal Disease

By Ruth L. Heller, DVM

When most people familiar with ferrets hear the term adrenal disease, they think of hyperadrenocortism. Ferrets with hair loss, enlarged prostates or vulvas, muscle and weight loss. Sometimes anemia, inability to urinate, and death. But there is another form of adrenal disease, and it is just as potentially deadly, if not more so.

Hyperadrenocorticism, sometimes referred to as Cushing's disease, is characterized by the overproduction of one or more of the adrenal hormones. In ferrets, those hormones are most often the sex hormones, estrogen and testosterone and their precursors. That overproduction leads to the clinical signs seen in affected ferrets, and can be caused either by hyperplasia (or overgrowth) of the adrenal tissue, or by neoplasia (cancer).

Hypoadrenocorticism, also known as Addison's disease, is the underproduction or complete absence of the adrenal hormones. Those hormones, in addition to the sex hormones and cortisol (or glucocorticoids), include those, called mineralocorticoids, that control the delicate balance of fluid and electrolytes within the body. When they are reduced or absent, that can lead to dehydration, low sodium and high potassium leading to muscular weakness and cardiac problems such as arrhythmias, anorexia, coma and if left untreated, death.

Addison's disease is not common in ferrets, and for that reason, many cases likely go undiagnosed. It can occur spontaneously, although that is rare (see case study in side-bar), but the main cause of Addison's disease is iatrogenic, or caused by removal of adrenal tissue by a surgeon treating hyperadrenocorticism. It is most commonly diagnosed based on clinical signs and bloodwork, with the hallmark of the disease being a reduced sodium:potassium ratio.

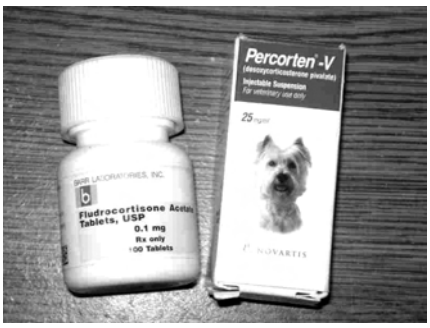
In some cases, a ferret with one very overactive adrenal gland will have a perfectly normal gland on the other side. When that happens, the normal gland will be suppressed, and will stop producing adrenal hormones. When the overactive adrenal is removed, it can take some time for the suppressed gland to begin working again. During that time, the ferret can develop clinical signs of Addison's disease. This will be temporary, and will resolve once the remaining adrenal is working normally, but will likely require treatment in the meantime.

In other cases, successful removal of both adrenal glands with no remaining accessory adrenal tissue will produce permanent Addison's disease. This must be treated for the remainder of the ferret's lifespan.

Treatment of Addison's disease involves replacing the missing mineralocorticoids, as well as the glucocorticoids. There are two treatment options available. The first is regular injections of Percorten to replace the mineralocorticoids, along with prednisone or another glucocorticoid. The second is daily administration of a drug called Florinef (generic name fludrocortisone), which may also require a glucocorticoid along with it, although Florinef has some glucocorticoid activity as well as mineralocorticoid activity.

I have successfully maintained Addisonian ferrets on both treatment

continued on page 22



PHOTOS BY DR. RUTH HELLER

Transient Hypoadrenocorticism in a Pregnant Jill

By Walton R. Dodson, Jr., DVM and Sally Heber

Summary

A two-year-old pregnant jill with signs and labwork consistent with a diagnosis of transient hypoadrenocorticism. Labwork and initial empirical treatment ruled out pregnancy toxemia.

The patient was ultimately treated, somewhat reluctantly because of her pregnancy, with predisone and Florinef. She recovered, delivered a healthy litter, was weaned off medication, and remains healthy and normal one year later.

Presentation

"Peggy Sue," a two-year-old dark sable jill was presented May 2, 2007, for extreme lethargy, inappetence and uncharacteristic behavior. She was bred April 8, 2007. Physical examination revealed slight dehydration, overall muscle weakness, normal cardiac and respiratory parameters. Her mucous membrane color and capillary refill time were normal. Her body temperature was 101.3 and continued to be normal throughout the course of her illness. Her kits were easily palpated abdominally.

We initially suspected pregnancy toxemia. A CBC and chemistry profile were submitted to a commercial laboratory. Pending lab results, Peggy Sue was treated with subcutaneous lactated ringers and calcium gluconate.

Initial lab results May 3 showed elevated potassium (6.6), low sodium (139), and slightly decreased hematocrit (29) and total protein (5.4). Dr. Dodson strongly suspected Addison's Disease (hypoadrenocorticism), but we were unable to find any reference to that disorder, other than cases occurring iatrogenically after surgical removal of both adrenal glands.

Because Percorten and dexamethasone as both strongly contraindicated in pregnancy, we began a dose of 1.25 mg of prednisone every 12 hours. The first dose produced no improvement. That evening, after further research, subcutaneous fluid treatment was changed to saline, and the dose of prednisone was reduced to .7 mg and she was again treated with subcutaneous calcium gluconate. She remained extremely weak (unable to walk or lift her head), but did eat, drink, urinate and defecate.

On May 4, Peggy Sue remained very weak. After discussion of possible consequences, she was treated with 0.025 mg Florinef (Fludrocortisone), 22 ml subcutaneous saline solution and 0.4 mg prednisone. The following day (May 5), we began a dosage regimen of 0.0125 mg Florinef and 0.4 mg prednisone every 12 hours on a continuing basis.

By May 6 she was lifting her head and she continued to eat and perform bodily functions. By May 8 she was able to use the litter pan on her own and move around on her own. By May 10 she was able to walk around the floor for a half hour. By May 15, she was close to normal energy and strength.

On May 19, without any assistance, she delivered a litter of seven healthy kits. She nursed all seven kits, and all grew to adult size without consequence.

Starting May 24, she was slowly weaned off the prednisone and Florinef. By June 1, 2007 she was no longer taking either medication. Bloodwork done June 1, 2007, showed electrolytes back within the normal range (sodium 143, potassium 4.7). Peggy Sue has remained healthy from that day forth. She was spayed September 6, 2007, without any adverse consequences.

PHOTOS BY SALLY HEBER

ADDISON'S *from page 10*

protocols. Percorten has the advantage of being an injectable, time release drug, while Florinef must be given every day and a single missed dose can prove fatal. The drawback to Percorten is that the interval between shots is variable for each ferret, and most ferrets have decreasing response to the drug, requiring it more often over time.

Currently, I give a single injection of Percorten at the time of surgery, if I suspect suppression of remaining tissue or removal of all adrenal tissue. The owner is instructed to give prednisolone if signs of Addison's disease develop. Since the initial duration of Percorten activity is between 21 and 28 days, electrolytes are checked at day 21, 25, and 30. If the ferret is clinically normal and bloodwork is normal, I recommend recheck at day 60. If not, treatment (Percorten or Florinef) is continued and regular bloodwork instituted.

I currently share my life with two Addisonian ferrets, both maintained on daily Florinef and prednisolone in their chicken gravy. Both of these ferrets became Addisonian after complete adrenalectomy, and both are doing very well. One of them is approaching three years post-surgery.

I recommend that owners of Addisonian ferret keep injectable saline fluids on hand and be comfortable with giving subcutaneous fluids in the event of clinical signs developing, and that they develop a close relationship with their veterinarian.

It is very possible to maintain these ferrets and give them normal, happy lives. The key is for both owner and veterinarian to be aware of the possibility of Addison's disease and to be aggressive about starting treatment. If this is done, a ferret with Addison's disease may never know that there is a problem.

Comparison of Percent Composition of Adult Domestic Mouse vs. Commercial Ferret Kibble (on a dry matter (DM) basis)

Food	Protein[1]	Fat[1]	Fiber[1]	Ash[2]	Carbs[2]	Moisture[2]
Adult domestic mouse	56.9%	23.5%	4.3%[3]	11.3%	4.0%[3]	67.4%
Natural Gold	55.6%	24.4%	5.6%	7.2%	7.2%	10.0%
Innova EVO	55.6%	23.3%	1.7%	9.4%	10.0%	10.0%
Mazuri	43.2%	23.3%	4.5%	8.5%	20.5%	12.0%
8-in-1 Ultimate (crunchy)	50.0%	17.8%	3.3%	7.8%[3]	21.1%[3]	10.0%
Zupreem	44.4%	22.2%	2.2%	7.8% [3]	23.4% [3]	10.0%
Totally Ferret (Adult)	40.0%	24.4%	1.7%	7.8%[3]	26.1%[3]	10.0%
Marshall Ferret Food	42.2%	20.0%	3.9%	7.2%	26.7%	10.0%

[1] not more than
[2] not less than
[3] information not listed; educated estimate

FERRET BIBLE

Researchers have recently uncovered a Bible written in the ferret language. They have managed to translate a small portion of the book of Genesis…

On the first day of creation, God created the ferret.

On the second day, God created hoomans to serve the ferret.

On the third day, God created all the animals of the earth to serve as potential play toys for the ferret.

On the fourth day, God created honest toil so that hoomans could labor to provide for the ferret.

On the fifth day, God created balls and tubes and plastic bags and stuffed animals and various other things so that the ferret might play with them for a few minutes before going back to destroying the belongings of their hoomans.

On the sixth day, God created veterinary science to keep the ferret healthy and the hooman broke.

On the seventh day, God tried to rest…but He had to clean the litterbox and all the corners.

– Author Unknown