## Panic Attack: Feline Hyperthyroidism

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If you have recently panicked—hyperventilating with your heart pounding in your throat—then you have experienced the magic of your sympathetic nervous system (SNS) at work. Also known as the fight-or-flight response system, its effects are universal throughout the animal kingdom. Now imagine those panic symptoms sustained and this may approximate the experience of the kitty (or human) with untreated hyperthyroidism.

Feline hyperthyroidism (FHT) is a disease that was initially reported in the late 1970's and is the most common feline metabolic disease. FHT is usually caused by nodules of hyperactive tissue that develop in the thyroid glands (which are located on each side of the trachea, commonly known as the windpipe). These nodules are typically benign but can be malignant (cancerous) in about 2% of FHT cases. Since the nodules of thyroid tissue are hyperactive, they produce excess amounts of thyroid hormone.

Thyroid hormone regulates the body's metabolism of carbohydrates, proteins, and fats. It also regulates body heat and regulates the SNS. The variety of body systems affected by thyroid hormone can consequently produce an array of clinical signs. However, the classic signs of the hyperthyroid cat are weight loss—particularly with maintained or even increased appetite—increased thirst/urination, vocalization, and increased activity. Cat owners often remark on their senior cat's youthful activity level or are pleased that their long-overweight cat has finally lost weight. More adverse-appearing signs can involve the GI tract (vomiting and diarrhea) or can produce undesirable behavioral changes (aggression). Hyperthyroidism can cause cardiovascular complications such as high blood pressure (which can, in turn, affect the heart, kidneys, and brain). As a result of its multi-organ effects, FHT is a life-threatening illness that requires treatment.

The standard method of diagnosing hyperthyroidism is a screening test that measures the total blood level of thyroid hormone, or total T4 (tT4). Sometimes a secondary blood test for a different format of thyroid hormone (free T4, or fT4) is also run. If hyperthyroidism is suspected but the results of both the tT4 and fT4 are inconclusive, additional tests may be considered, such as a T3 suppression test, or radioactive scintigraphy, which targets and captures images of the hyperactive thyroid tissue.

If a test result is consistent with hyperthyroidism, there are treatment options to manage or cure the disease. The most common initial treatment recommended is a medication, methimazole, which is available in oral or transdermal (absorbed through the skin) formulations and has an approximately 95% response rate. This is a medication that will be required by the hyperthyroid patient for life, or until either I-131 or thyroidectomy are performed (more to follow on these). The patient receiving methimazole will require routine blood thyroid level and kidney value monitoring to determine appropriate dosing—it is likely that the dose the patient requires will increase over time, as the thyroid nodules can continue to grow.

The treatment of choice for FHT is a dose of radioactive iodine, or I-131, which specifically targets the hyperactive thyroid tissue. This treatment has an approximately 95% cure rate and is typically performed at a specialized facility. There is the potential, with this treatment, to cause hypothyroidism (wherein the thyroid gland cannot generate enough thyroid hormone), which would require thyroid supplementation for the remainder of the patient's life.

Thyroidectomy, or the surgical removal of the thyroid glands, has an approximately 90% cure rate when both thyroid glands are removed. Complications of this procedure include those of any surgical procedure requiring anesthesia in addition to inadvertent removal of the parathyroid glands (these reside near the thyroid glands), which regulate the body's calcium levels.

Another treatment is that of a prescription iodine-reduced diet (thyroid hormone incorporates iodine into its structure). This treatment has an approximately 82% response rate as long at the patient is ingesting this diet exclusively. It has also been shown to be safe for patients with concurrent kidney disease. Difficulties arise with patients who are picky (and reject the prescription food) or who cannot be limited to only the prescription diet.

Regardless of the treatment selected, routine monitoring of the patient's bloodwork through the remainder of her/his life is recommended—particularly as treating hyperthyroidism can unmask underlying kidney disease.

## References

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