Hyperthyroidism in Cats

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Introduction

About 10%[1] of cats over 10 years of age suffer from hyperthyroidism. The thyroid consists of two glands that sit on either side of the trachea. The thyroid glands are regulated by the autonomic nervous system – the hypothalamus releases thyrotrophin-releasing hormone, which acts on the pituitary gland, which in turn releases thyroid-stimulating hormone, which acts on the thyroid glands. They release thyroid hormone, which is made of tyrosine and iodine, and work on a negative feedback loop. Thyroid hormone increases metabolism, heart rate, oxygen consumption, muscle and adipose catabolism, and catecholamine response; glucocorticoids like prednisolone inhibit active thyroid hormone.

In hyperthyroidism, adenomatous hyperplasia occurs in the thyroid glands, increasing their size, resulting in a palpable goitre on the neck. The exact etiology is unknown, but one cause is a lack of iodine in the diet, causing the thyroid to work overtime to collect as much iodine from the diet as it can. This can result in growth of the gland and, with an increase of iodine in the diet, hyperthyroidism.

When diagnosing hyperthyroidism in a cat, consider the fact that thyroid hormone typically plummets in sick animals (Sick Euthyroid Syndrome). Additionally, most hyperthyroid cats will also have concurrent diseases, including cardiomyopathy, chronic kidney disease (CKD), and cardiomyopathy. Be aware that hyperthyroid can mask the signs of CKD because of the increase in metabolic rate (which increases glomerular filtration rate in the kidneys), which allows the kidneys to continue working even though the cells are starting to fail.

Signalment

Cats >10 years old[3] (median 12-13)[1,4]

Purebreed cats are less at risk, especially Siamese and Himalayan breeds

Clinical Signs

This disease develops slowly, so it is often picked up on a routine clinical exam/bloodwork.

Main sign: ravenous plus weight loss

Possible clinical signs and their reasons are listed below:

- Polyphagia/Polydipsia/Polyuria (because increased metabolic rate, so eating, drinking, urinating more)
- Weight loss (increased lipid metabolism)
- Goitre (beware of larynx (hard structure) and don't stretch head too much; could also be a cyst; adenomatous hyperplasia from lack of iodine)
- Hypertension (increased metabolism; may lead to blindness)
- Cold seeking behavior (increased metabolism makes them hot)
- Tachypnea/dyspnea/panting (increased metabolism = increased water and CO_2 -> urinate water, breathe faster to get rid of CO_2)

- Tachycardia/gallop/murmur (increased metabolism, can lead to cardiac failure)
- Hyperactive/Irritable/Restless (high metabolic rate -> go, go, go), increased vocalization
- Vomiting and Diarrhea (from eating too much too fast; diarrhea also because of decreased Cobalamin (B_{12}))
- Severe muscle weakness, seizures (decreased B_{12}/Cobalamin)
- Poor coat (too busy to groom)

Hematology/Biochemistry

- Cholesterol/triglycerides decrease (metabolism)
- Hyperglycemia (stress, increased carbohydrate metabolism, so leaves more glucose in the blood)
- Increased Red blood cells (RBCs; erythropoiesis)
- Elevated liver enzymes (ALT, ALP) – thyroxine is hepatotoxic and liver working overtime
- Hyperphosphatemia (increased bone metabolism)
- Urinalysis – specific gravity down, Protein: creatinine down, prone to UTIs
- Situational hypertension

Diagnosis

1) Start with Total T_4 test – increase
2) If Total T_4 is not definitive, test Free T_4 – increase
    a. More sensitive, but can result in more false positives, so always run after total T4

*Remember, if there is concurrent disease, T4 may be high normal (Sick Euthyroid Syndrome lowers T4, so it won't appear high)

Other options:

3) Scintigraphy
4) cTSH (canine thyroid-stimulating hormone) assay (difficult to differentiate mild hyperthyroid from euthyroid)
5) TRH (thyrotropin-releasing hormone) stimulation test
6) T_3 suppression test

If diagnosis is not definitive yet, a wait and watch approach should be adopted. The disease is slow moving, so waiting 2-3 months and retesting total T_4 is better than misdiagnosing the disease and causing hypothyroid.

Treatment

Medical Management:

1) Diet – low iodine (Hill's y/d)
    a. Good option to start, but it doesn't taste great, so cat's are less likely to eat it when the thyroid is more under control
2) Medication – Methimazole, Carbimazole (breaks down into Methimazole), Thiamazole
    a. Block thyroid hormones, but the thyroid continues to grow and problem can worsen over time

b. Adverse effects:
    i. GI signs – switch to Carbimazole or trans-dermal gel (wear gloves!!)
    ii. Any other – withdraw meds and manage another way

Recheck 2-3 weeks after diagnosis – T4, urea/creatinine, liver values, hematology, blood pressure, urinalysis. Once stable, consider long-term/permanent options. Ongoing monitoring every 3-6 months.

Permanent Treatments (only after thyroid is under control – may not be suitable with concurrent disease, ie CKD):

1) Radioactive Iodine (I^{131})
    a. Taken up by the thyroid looking for iodine and nukes the cells
2) Surgery – Thyroidectomy (be careful not to take the parathyroids as well)
    a. Complications: recurrence (less if bilateral), laryngeal paralysis, hypocalcemia, hypothyroidism
    b. Supplement with Calcium and Vitamin D afterwards

Methimazole plus I131 has the best results. Long term prognosis is good, but requires regular management and blood tests to check for renal (kidney) disease

References

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