Unilateral Extracapsular Thyroidectomy for a Non-Functional Cystic Thyroid Adenoma

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CASE HISTORY

An 11-year-old, female neutered, Siamese cat presented with a subcutaneous right-sided ventral neck mass as an incidental finding during treatment for bacterial cystitis. An enlarged thyroid gland was suspected but there were no clinical signs of thyrotoxicosis.

CLINICAL EXAMINATION

The cat was initially presented with a chronic history of bacterial cystitis. The neck mass was approximately 2cm long and 0.5cm wide, and situated on the right side of the ventral neck caudal and adjacent to the larynx. Clinical signs consistent with thyrotoxicosis, such as restlessness, tachycardia, renal or gastro-intestinal abnormalities, were not evident. There were no abnormalities on serum biochemistry and haematology profiles. The initial serum thyroxine level of 41 nmol/L was within normal laboratory limits. A fine needle aspirate of the mass yielded one millilitre of a yellow, translucent, viscous material. A lipoma was diagnosed on the basis of cytological analysis of the aspirate by the laboratory. No abnormalities were detected on thoracic radiographs in left lateral, right lateral, and ventrodorsal projections. Serum thyroxine analysis was performed every three months and all results were within normal laboratory parameters.

The cat presented 12 months later for the treatment of a right-sided carnassial tooth root abscess. The subcutaneous mass in the neck had approximately doubled in size and an occasional cough was present. A repeat serum thyroxine level was within normal limits. Respiration was normal with no inspiratory effort or stridor evident. The cat was admitted for surgical excision of the ventral cervical mass.

TREATMENT

The cat was premedicated with acepromazine (0.04mg/kg SC) and general anaesthesia was induced with and maintained on gaseous isoflurane and oxygen. She was placed in dorsal recumbency, the head hyperextended with a rolled towel placed beneath the neck, and the thoracic limbs pulled caudally. An intravenous infusion of a balanced electrolyte solution (10ml/kg/hr) was administered during surgery.

The mass was approached with a ventral midline incision from the larynx to manubrium. The paired sternohyoideus muscles were divided by blunt dissection and retracted with Gelpi self-retaining retractors. The mass was identified as an...
enlarged right thyroid gland. The left thyroid gland appeared grossly normal.

The right thyroid gland was excised using a modified extracapsular technique. The internal jugular vein, carotid artery, vagosympathetic trunk, and recurrent laryngeal nerve were identified. The cranial thyroid artery, distal to the parathyroid artery, was double ligated with 4-0 polyglyconate suture material. The capsule of the thyroid gland was sharply incised around the external parathyroid gland and the remaining capsule and thyroid gland removed with a saline-moistened cotton-wool bud. The sternohyoideus muscles were closed with 3-0 polyglyconate in a simple continuous pattern. The skin was closed with 3-0 nylon in a interrupted cruciate pattern.

Post-operatively, the cat was maintained on intravenous isotonic saline (60ml/kg/day) supplemented with potassium chloride (15mEq/L). Serum calcium levels were assayed for 48 hours following surgery and were within normal limits.

DIAGNOSIS AND RESULTS
Histopathological examination of the submitted mass gave a diagnosis of cystic thyroid adenoma.

Serum thyroxine levels performed three, six, nine, 12 and 24 months post-operatively were within normal limits. The left thyroid gland was not palpably enlarged and there were no clinical signs consistent with thyrotoxicosis during this period.

DISCUSSION
The diagnosis of feline hyperthyroidism is based on history, clinical signs, physical findings, blood tests, and radionucleide scans (Peterson, 1995). The most significant clinical finding is a palpable enlargement in one or both thyroid glands as normal thyroid glands are not palpable (Peterson, 1995). The failure to palpate thyroid glands may result from ventral descent of the thyroid glands into the thoracic inlet or adenomatous or malignant changes in ectopic tissue (Peterson, 1995). Thyroid glands may be palpable without clinical or laboratory evidence of hyperthyroidism but these adenomatous changes will become functional (Peterson, 1995). Functional hyperthyroidism was not evident in this case despite a three-year history of palpable thyroid gland enlargement.

There are different histopathological types of thyroid gland adenomas. The follicular adenoma may expand through progressive cystic degeneration. These cystic adenomas contain one to two large cavities filled with proteinaceous fluid, necrotic debris, and erythrocytes. They are surrounded by a capsule of dense connective tissue with focal accumulations of tumour cells (Capen, 1993).

Feline hyperthyroidism is a multisystemic disorder resulting from excessive circulating concentrations of triiodothyronine (T3) and thyroxine (T4) (Jones, 1993; Watt, 1994; Peterson, 1995). Hyperthyroidism is the most common endocrine disorder of middle to older aged cats.
between four and 22 years of age (Peterson, 1995). A breed or sex predilection has not been identified but some have reported an increased incidence in domestic medium and long-haired cats (Peterson, 1995; Mooney, 1996). Hyperthyroidism in cats is caused by functional benign thyroid adenomas or adenomatous hyperplasia of the thyroid glands (Watt, 1994; Peterson, 1995; Peterson & Becker, 1995). Thyroid adenocarcinoma is a rare cause of feline hyperthyroidism (Watt, 1994; Peterson, 1995).

The clinical signs associated with hyperthyroidism result from the stimulatory effects of excessive thyroid hormones on various organ systems (Peterson, 1995). The clinical severity varies from mild to marked depending on the duration of hyperthyroidism, the presence of concurrent diseases, and the ability to meet the increased demands imposed by excessive thyroid hormone secretion (Peterson, 1995). The benign thyroid adenoma in this case was considered non-functional as clinical signs and laboratory evidence of thyrotoxicosis were not evident. Laryngeal hemiparesis in a cat has been reported to result from peripheral compression of the recurrent laryngeal nerve by a functional cystic thyroid adenoma (Rozanski & Stobie, 1995). The cat in this case report presented with a cough due to laryngeal compression or irritation but there was no evidence of laryngeal paralysis.

Diagnosis of feline hyperthyroidism depends on the demonstration of increased serum levels of thyroid hormone concentrations (Peterson, 1995). Resting T3 and T4 levels are above normal ranges. Five to 10 percent of cats have normal T3 levels with elevated T4 levels and are usually associated with mild clinical signs of hyperthyroidism (Peterson, 1995). Normal serum thyroxine concentrations can occur in hyperthyroid cats as T4 levels can fluctuate in and out of normal laboratory parameters with mild hyperthyroidism and concurrent non-thyroid illness may suppress serum thyroxine levels to the high normal range (Jones, 1993; McLoughlin et al., 1993). Provocative tests, using exogenous thyrotropin-releasing hormone or thyroid-stimulating hormone (TSH), are being investigated as a means of diagnosing hyperthyroidism with borderline T4 blood levels (Jones, 1993; Peterson, 1995). The T3 suppression test may be a more reliable method for confirming hyperthyroidism (Peterson, 1995). The administration of exogenous T3 will normally result in a negative feedback on the hypothalamus-pituitary-thyroid axis causing inhibition of TSH release and decreased endogenous thyroid hormone secretion (Peterson, 1995). Thyroid hormone secretion is autonomous and independent of TSH regulation in hyperthyroid cats. Hence, exogenous T3 will not affect serum thyroid hormone concentrations (Peterson, 1995). In this case, serum thyroxine levels were within normal limits but a provocative or T3 suppression test may have demonstrated subclinical hyperthyroidism.

Haematological changes with functional feline hyperthyroidism include a mature leucocytosis and neutrophilia, eosinopaenia, and a mild to moderate erythrocytosis (Peterson, 1995). Biochemical abnormalities include elevations in alkaline phosphatase, alanine transferase, aspartate aminotransferase, blood urea nitrogen, and creatinine (Peterson, 1995). Biochemical and haematological abnormalities were not detected in this case.

Radionucleide imaging of the thyroid glands is a useful technique for the diagnosis of hyperthyroidism but was not readily available in this case. Radionucleide imaging also determines the degree of involvement and location of abnormal tissue, the presence of ectopic tissue, and the detection of regional and distant metastases (Peterson, 1995).

The treatment of feline hyperthyroidism, which aims at controlling excessive thyroid hormone secretion from hyperfunctioning adenomatous tissue, involves either chronic antithyroid medical therapy, surgical thyroidectomy, or radioactive iodine administration (Jones, 1993; Peterson, 1995; Peterson & Becker, 1995; Mooney, 1996). The therapeutic options depend on age, concurrent cardiovascular and renal disease, the availability of nuclear medicine facilities, the skill of the surgeon, and owner compliance (Watt, 1994). Surgical treatment was selected in this case because of extraluminal compression or irritation of the cervical mass of the larynx and not functional hyperthyroidism.

Surgical treatment of hyperthyroidism is effective and relatively simple, but can be associated with significant morbidity and mortality. Thyroidectomy is curative and rapidly normalises serum thyroxine concentrations within 48 hours (Peterson, 1995). The disadvantages of thyroidectomy include anaesthetic risk, difficult intra-operative assessment of abnormal tissue, a 9% mortality rate, and post-surgical complications such as hypoparathyroidism, recurrence of hyperthyroidism, and hypothyroidism (Peterson, 1995).

The selection of premedication and anaesthetic agents is important as the effects of age and cardiac and metabolic abnormalities are significant risks (Peterson, 1995). Premedication with acepromazine is recommended as it reduces the autonomic manifestations of hyperthyroidism and prevents arrhythmias induced by thiobarbituates and inhalation agents (Birchard, 1994; Flanders, 1994; Watt, 1994; Peterson, 1995). Xylazine potentiates cardiac arrhythmias. Anticholinergic agents, such as atropine, stimulate adrenergic activity and result in tachycardia and arrhythmias (Peterson, 1995). Ketamine increases sympathoadrenal activity and may cause catecholamine-induced arrhythmias but some recommend ketamine as an induction agent (Peterson, 1995).

Intravenous thiobarbituates or isoflurane are preferred for anaesthetic induction as they have antithyroid activity but thiobarbituates are arrhythmic and should be administered carefully in emaciated cats (Peterson, 1995). Isoflurane is recommended for maintenance of anaesthesia as halothane and methoxyfluorance sensitise the heart to catecholamine-induced arrhythmias (Flanders, 1994; Peterson, 1995). Intra-operative ECG and anaesthetic monitoring is recommended as ventricular arrhythmias are common in hyperthyroid cats (Birchard, 1994; Flanders, 1994; Peterson, 1995).

Thyroidectomy techniques include intracapsular, modified intracapsular, extracapsular, modified extracapsular, and staged bilateral procedures (Flanders, 1994; Watt, 1994; Peterson, 1995). The aim of thyroidectomy is to remove abnormal thyroid tissue and preserve at least one parathyroid gland (Peterson, 1995). Bilateral adenomatous hyperplastic tissue is identified in 70% of functional
hyperthyroidism due to retained remnants of hypoparathyroidism is reduced but complications include period post-operatively revealed a euthyroid cat in this case. Hyperthyroidism following unilateral thyroidectomy is observed within 12 months in the contralateral thyroid gland (Peterson, 1995). Regular clinical examinations and analysis of serum thyroxine concentrations for a 24-month period post-operatively revealed a euthyroid cat in this case.

The intracapsular techniques preserve the thyroid capsule and external parathyroid gland. Hence the risk of hypoparathyroidism is reduced but complications include recurrent hyperthyroidism due to retained remnants of normal thyroid tissue (Birchard, 1994; Flanders, 1994; Watt, 1994; Peterson, 1995; Mooney, 1996). The extracapsular techniques remove the thyroid gland and its capsule but complications include hypoparathyroidism (Peterson, 1995; Mooney, 1996). If the parathyroid gland viability is compromised, then the gland should be minced and reimplanted into adjacent muscle to permit revascularisation and possible return to normal function (Jones, 1993; Flanders, 1994; Peterson, 1995). Some authors recommend bilateral thyroidectomy, regardless of gross size or appearance, to avoid further surgical intervention (Watt, 1994; Peterson, 1995).

Bilateral thyroidectomy will result in hypothyroidism which requires thyroxine administration although thyroid levels may return to normal (Birchard, 1994; Watt, 1994; Peterson, 1995). Thyroxine supplementation is rarely required following unilateral thyroidectomy despite subnormal T3 and T4 concentrations (Peterson, 1995). Serum T4 concentrations should be reassessed annually (Peterson, 1995). Recurrence of hyperthyroidism is caused by the inability to remove all the abnormal thyroid tissue, failure to recognise abnormal tissue, the presence of either abnormal ectopic tissue or metastases, and redevelopment of hyperplastic adenomatous tissue in the remaining thyroid gland (Flanders, 1994; Watt, 1994; Peterson, 1995). Medical or radioactive iodine therapy is recommended for the treatment of recurrent hyperthyroidism (Peterson, 1995).

Hypocalcaemia is the most significant complication of thyroidectomy (Jones, 1993; Birchard, 1994; Flanders, 1994; Graves, 1994; Watt, 1994; Peterson, 1995; Mooney, 1996). Serum calcium levels should be monitored following surgery, especially if bilateral thyroidectomy has been performed (Jones, 1993; Peterson, 1995). Hypocalcaemia occurs most commonly one to three days following surgery with clinical signs including restlessness, weakness, muscle tremours, twitching, tetany, and seizures (Jones, 1993; Birchard, 1994; Flanders, 1994; Graves, 1994; Watt, 1994; Peterson, 1995). The treatment includes intravenous calcium gluconate, and oral calcium and vitamin D (Jones, 1993; Graves, 1994; Watt, 1994; Peterson, 1995). Spontaneous recovery from hypoparathyroidism can occur through reversible damage or hypoxia resulting from thyroidectomy or compensation by accessory parathyroid tissue (Peterson, 1995). Other complications include laryngeal paralysis and Horner’s Syndrome resulting from intra-operative damage to the recurrent laryngeal nerve and cervical sympathetic trunk respectively (Graves, 1994; Peterson, 1995).

Radioactive iodine therapy is a simple, effective, and safe method for the treatment of functional feline hyperthyroidism (Craig et al., 1993; Jones, 1993; Peterson, 1995, Peterson & Becker, 1995). The goal of radioactive iodine therapy is to restore euthyroidism and prevent hypothyroidism with a single injectable or oral dose of radiation (Peterson, 1995; Peterson & Becker, 1995). The radioisotope 131I concentrates primarily in hyperplastic and neoplastic thyroid cells where subsequent irradiation results in destruction of all hyperplastic and neoplastic tissue including ectopic and metastatic tissue (Peterson, 1995). Seventy-five percent of cases are euthyroid within eight days of a single intravenous treatment (Peterson, 1995).

Radioactive iodine can be administered by intravenous, subcutaneous, or oral routes (Mooney, 1994; Peterson, 1995, Peterson & Becker, 1995; Mooney, 1996). Response to radioactive iodine is reduced by high serum thyroxine concentrations, large thyroid glands, and anti-thyroid drug administration (Jones, 1993). The disadvantages of radioactive iodine treatment include limited availability of nuclear medicine facilities, radiation safety regulations, expense, prolonged hospitalisation, and difficulty in determining correct dose (Peterson, 1995).

REFERENCES


