VETERINARY CLINICS
SMALL ANIMAL PRACTICE

Feline Thyroid Storm

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*T*hyrotoxicosis is a term used to describe any condition in which there is an excessive amount of circulating thyroid hormone, whether from excess production and secretion from an overactive thyroid gland, leakage from a damaged thyroid gland, or an exogenous source. In most veterinary patients, thyrotoxicosis occurs from thyroid gland hyperfunction. Feline hyperthyroidism is a common endocrinopathy in middle-aged to older cats and is most often the cause of thyrotoxicosis seen by veterinarians. Although less common, active thyroid carcinomas in cats and dogs can also result in severe thyrotoxicosis. The clinical presentation of thyrotoxicosis in veterinary patients can vary tremendously from asymptomatic biochemical changes to life-threatening multisystemic disease. In human beings, one form of acute thyrotoxicosis is called thyroid storm and is a cause of significant mortality in human emergency rooms. Thyroid storm is uncommon, and the signs can go unrecognized, thus contributing to the high degree of mortality associated with this disease. In human beings, thyroid storm can occur at any age. It can be present in euthyroid patients as well as in treated and partially treated hyperthyroid patients.

Although thyroid storm is a rare but well-recognized syndrome in human medicine, it has not been described as a clinical entity in veterinary medicine. Most frequently, acute thyrotoxicosis is diagnosed in hyperthyroid cats, although dogs with functional carcinomas or after accidental oversupplementation with thyroid hormone are also presented. As with human medicine, early recognition of acute thyrotoxicosis and aggressive therapy can improve the clinical outcome of such patients.

PATHOGENESIS
Just what precipitates the actual thyroid storm syndrome in certain thyrotoxic patients is unknown [1]. Because multiple factors seem to be involved, the exact pathogenesis of the disease is even more clouded. Thyroid hormone causes a cellular effect by the free hormone diffusing into the cell and binding to response elements in the nucleus. The result is thyroid hormone–specific gene expression, resulting in altered cellular metabolism. Therefore, the

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availability of free thyroid hormone would seem to be an important part of the pathogenesis of thyroid storm.

Initially, one might surmise that circulating thyroid hormones would be significantly higher in patients with thyroid storm than in other thyrotoxic patients. Early studies in thyrotoxic human patients attempted to show such a difference, and total and free thyroid hormone levels were compared between patients with thyroid storm and uncomplicated hyperthyroid patients. Some isolated case reports did show transient elevations in free hormone or changes in thyroxine (T_4)-binding globulin levels in patients with thyroid storm [2]. These biochemical parameter changes are consistent with the presence of nonthyroidal illness, however, and because nonthyroidal illness is a known precipitant of thyroid storm, they may not be diagnostic of thyroid storm itself [3,4]. Further studies have shown that there is no difference between serum total or free thyroid hormone levels in patients with thyroid storm and in more stable hyperthyroid patients in human medicine [5,6].

The rapidity and magnitude of change in the serum thyroid hormone level may be more important than the actual serum levels themselves. This would explain the occurrence of thyroid storm after radioactive iodine therapy and thyroidal surgery, both of which potentially damage the thyroid gland, causing rapid release of hormone [7]. Also supporting this theory is that thyroid storm has been reported to follow abrupt cessation of antithyroid medication or accidental thyroid hormone overdose, both resulting in the rapid rise of serum thyroid hormone levels [8,9]. Additionally, nonthyroidal illness is known to be a precipitating factor for thyroid storm in human medicine. Nonthyroidal illness has been shown to alter binding of thyroid hormones to their carriers. Changes in thyroid hormone–binding protein affinity could be responsible for a rapid increase of circulating free thyroid hormone available to activate cellular targets [10]. A sudden increase of inappropriately activated cells by thyroid hormone could certainly result in thyroid storm.

Activation of the sympathetic nervous system has been implicated in the onset of thyroid storm [11]. Evidence supporting this is that many of the clinical signs and physiologic symptoms seen in thyroid storm are similar to those seen during catecholamine excess. Additionally, medical adrenergic blockade can dramatically reduce clinical signs seen with thyroid storm. In human beings, it has been shown that serum and urine catecholamine levels are within normal limits during thyroid storm [12]. It is known that thyroid hormones can alter tissue sensitivity to catecholamines, however. This can occur at the cell surface receptor as well as at the intracellular signaling levels, and this increased sensitivity may result in the clinical signs seen during thyroid storm [13]. Beta-blockade does not completely prevent thyroid storm [14], however, although it may ameliorate some of the clinical signs. These findings lead to the conclusion that factors other than activation of the sympathetic nervous system are probably important in the development of clinical signs associated with thyroid storm.

There is some evidence that thyroid storm not only results from relative increases in circulating thyroid hormone but that cellular response to thyroid

hormone may be enhanced. This effect has been implicated in the cause of thyroid storm resulting from infection, sepsis, hypoxemia, hypovolemia, and lactic or ketoacidosis [15]. Similar enhanced cellular responses may be present in thyrotoxic veterinary patients. In hyperthyroid cats, increased serum concentrations of cardiac troponin I, a marker of cardiac myocyte injury, have been demonstrated [16]. Successful treatment of the hyperthyroidism and reduction of the serum T_4 levels resulted in a decrease of the troponin. Additionally, thyroid hormone has been shown to increase Na^+ current and intracellular Ca^{++} in isolated feline atrial myocytes [17]. These data suggest that exposure to excess thyroid hormone may directly result in alteration of cellular response in the cat or at least in feline cardiac myocytes.

PRECIPITATING EVENTS

In most cases of thyroid storm in human beings, a precipitating event can be identified, although no known causes are found in up to 2% of cases [18]. The most common events are infection, thyroidal and nonthyroidal surgery, radioactive iodine therapy, administration of iodinated contrast dyes, administration of stable iodine, withdrawal of antithyroid medication, amiodarone therapy, ingestion of excessive amounts of exogenous thyroid hormone, vigorous palpation of the thyroid, severe emotional stress, and a variety of acute nonthyroidal illnesses. Common events that may precipitate thyroid storm in feline hyperthyroid patients include radioactive iodine therapy, thyroidal surgery [19], or vigorous thyroid palpation causing destruction of thyroid cells and release of thyroid hormone into the circulation (Box 1). Abrupt withdrawal

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Box 1: Potential precipitating factors for feline thyroid storm

Associated with acute increase in circulating thyroid hormones

Abrupt withdrawal of methimazole or antithyroid medication

Iodine 131 therapy

Thyroidal surgery

Palpation of the thyroid

Administration of stable iodine compounds

Inappropriate ingestion of excessive thyroid hormone supplementation

Associated with nonthyroidal illness

Stress

Infection

Nonthyroidal surgery

Trauma

Thromboembolic disease

Vascular accidents

of antithyroid medication could result in an acute elevation of circulating thyroid hormone, as could the administration of stable iodine compounds, which result in an initial increase of thyroid hormone synthesis in the cells. Stress and nonthyroidal illness, especially infections, are most likely important for progression of the clinical course in hyperthyroid cats to thyroid storm. The presence of any of the other causes found as precipitating factors in human beings could also play a role in the precipitation of thyroid storm.

CLINICAL SIGNS

Thyroid storm is the acute exacerbation of clinical signs of thyrotoxicosis; however, the diagnosis of thyroid storm in human medicine is primarily a clinical one. In human beings, it is based on the prevalence of four major clinical signs. These include fever; central nervous system (CNS) effects ranging from mild agitation to seizures or coma; gastrointestinal-hepatic dysfunction ranging from vomiting or diarrhea and abdominal pain to unexplained jaundice; and cardiovascular effects, including sinus tachycardia, atrial fibrillation, and congestive heart failure. The combination of these clinical signs, along with identification of a precipitating event, allows for the diagnosis of thyroid storm [18]. In cats presenting with presumed thyroid storm, many of these clinical signs also occur (Box 2). Such cats often show mild to severe respiratory distress. Auscultation may reveal a cardiac murmur or arrhythmia, most often a gallop rhythm [20]. Crackles or dullness in the lung fields indicating pulmonary edema or pleural effusion, respectively, associated with congestive heart failure may also be auscultated [21]. Additional clinical signs that may be associated with thyroid storm in cats include mild to severe hypertension [22]. Retinopathies, including hemorrhage, edema, degeneration, or even retinal

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Box 2: Clinical signs of feline thyroid storm

Tachypnea
Tachycardia
Hyperthermia
Respiratory distress
Cardiac murmur
Cardiac arrhythmia
Auscultatory crackles or dullness
Sudden blindness
Severe muscle weakness
Ventroflexion of the neck
Absent motor limb function
Neurologic abnormalities
Sudden death

detachment, may be found, especially in hypertensive thyrotoxic cats [23]. Tachypnea and hypothermia may be present, and absent limb motor function may be detected as a result of thromboembolic disease occurring from acute thyrotoxicosis [24]. Severe acute muscle weakness and ventroflexion of the neck may be seen in acutely thyrotoxic cats, often associated with hypokalemia [25]. Cats in thyroid storm may exhibit a myriad of neurologic abnormalities ranging from hyperexcitability to stupor [26]. Sudden death may also occur.

DIAGNOSIS

The diagnosis of thyroid storm is based on identification of the presence of thyrotoxicosis, appropriate clinical signs, and evidence of a precipitating event [18]. Thyrotoxicosis in hyperthyroid cats is demonstrated by an elevated total T_4 level or a total T_4 level in the high normal range combined with an elevated free T_4 level or with lack of suppression by triiodothyronine (T_3) [27]. In some cases, the total T_4 level may be in the normal range in a hyperthyroid cat, but in cases of thyroid storm, the total T_4 and free T_4 levels are expected to be higher than the reference range. The severity of clinical signs in hyperthyroid cats does not seem to correlate with the absolute level of circulating thyroid hormone. Therefore, as in people, the diagnosis of thyroid storm in cats probably cannot be based on absolute serum thyroid hormone levels. In human medicine, thyroid storm is diagnosed based on a point system assigned to each of the main clinical components: fever, CNS signs, gastrointestinal signs, and cardiovascular signs as well as the presence or absence of a precipitating event [18]. In hyperthyroid feline patients, thyroid storm may be diagnosed based on the presence of clinical signs of acute thyrotoxicosis, as described in the preceding paragraph. The owners should be questioned and the clinical case reviewed thoroughly to identify a precipitating event. If one can be found, it would further narrow the diagnosis to thyroid storm.

LABORATORY ABNORMALITIES

Laboratory abnormalities are those seen resulting from uncomplicated thyrotoxicosis [20,28]; there is no distinguishing laboratory value(s) for the diagnosis of feline thyroid storm. In the hyperthyroid cat, hematologic abnormalities may include mild erythrocytosis, macrocytosis, and Heinz body formation. In human patients with thyroid storm, leukocytosis with a left shift in the absence of active infection or inflammation has been identified [29]. In hyperthyroid cats, mature neutrophilia, lymphopenia, and eosinopenia are more commonly identified as stress responses. Biochemical abnormalities seen in people with thyroid storm include mild hyperglycemia and hypercalcemia. Elevated liver enzymes are seen as well, and hyperbilirubinemia may occur in severe cases. This finding carries a poor prognosis. In hyperthyroid cats, elevated liver enzymes, mild hyperglycemia, hyperbilirubinemia, and severe hypokalemia may be seen in acute thyrotoxicosis. A decreased sodium/potassium ratio may be seen in thyrotoxic cats that are presented in heart failure with pleural effusions [30]. Mild to severely elevated creatine kinase may be seen in cats

presenting with thyroid storm. Radiographs may reveal an enlarged heart or evidence of congestive heart failure. Echocardiography may show hypertrophy of the left ventricular wall or left interventricular septum [31]. Myocardial contractility deficits also may be seen.

TREATMENT

Treatment of thyroid storm is aimed at controlling the four major problematic areas: (1) to reduce the production or secretion of thyroid hormones, (2) to counteract the peripheral effects of thyroid hormones, (3) to provide systemic support, and (4) to identify and eliminate the precipitating factor [32].

Reduction in the Production or Secretion of New Thyroid Hormones

The thioimidazole compound methimazole inhibits iodine incorporation into tyrosyl residues of thyroglobulin, and thus prevents the synthesis of active thyroid hormone. As a result, methimazole should be the first line of defense against thyroid storm. It does not prevent the secretion of already formed thyroid hormones, however. Methimazole may be given orally, transdermally, or even rectally in cats [33]. The dose should be toward the high end (5 mg administered twice daily) in cats that have normal renal function [34]. If there is suspected renal insufficiency or failure, the dose of methimazole should be reduced by half.

Methimazole blocks the formation of new active thyroid hormone, but other therapy must be instituted to prevent further secretion of formed hormone, which is stored in high concentrations in the thyroid gland. This can be done by treatment with stable iodine compounds, such as potassium iodine. In large doses, these compounds can also decrease the synthesis rate of thyroid hormone. They must be given 1 hour after methimazole administration, because a large load of iodine initially stimulates thyroid hormone production. Potassium iodate, a more stable form of potassium iodine, has been used successfully in cats and may be given at a dose of 25 mg every 8 hours [35]. Instead of potassium iodide, lipid-soluble radiographic contrast agents containing stable iodine, such as iopanoic acid, may be given. Such compounds have been used in hyperthyroid cats as an ancillary treatment for hyperthyroidism. Iopanoic acid or diatrizoate meglumine may be given at a dose of 100 mg by mouth twice daily [36]. Although iopanoic acid is available in a parenteral form, oral dosing is safer because it is a hyperosmolar agent. These compounds have the additional advantages of blocking peripheral conversion of T_4 to T_3, blocking T_3 binding to its receptor, and inhibiting thyroid hormone synthesis [37].

Inhibition of Peripheral Effects of Thyroid Hormone

The most rapid relief of signs caused by thyroid storm is by medications that block the β-adrenergic receptors, such as propranolol and atenolol. The nonselective beta-blocker propranolol, most commonly used as a sympatholytic in human medicine, is inherently difficult to use in cats because of its poor oral bioavailability and short half-life, requiring dosing every 8 hours. Its use has been largely superseded by that of atenolol because of its selectivity and the

once-daily dosing regimen [38]. Propranolol has been shown to inhibit the peripheral conversion of T_4 to T_3, although this effect happens slowly [39]. Therefore, its use may be advantageous in severely thyrotoxic cats [35]. Additionally, it may be used intravenously. Propranolol should be used toward the high end of the dose range at 5 mg administered by mouth every 8 hours or 0.02 mg/kg administered intravenously over 1 minute to ensure β-adrenergic blockade. Alternatively, the selective β$_1$-adrenergic blocker atenolol may be used at a dose of 1 mg/kg administered every 12 to 24 hours. In acute situations, the short-acting β$_1$-adrenergic blocker esmolol may be used intravenously at a loading dose of 0.5 mg/kg administered intravenously over 1 minute, followed by a constant rate intravenous infusion of 10 to 200 μg/kg/min.

An extreme method to fight the peripheral actions of excess thyroid hormones is to reduce the systemic levels present. Peritoneal dialysis, plasmapheresis, and hemodialysis have been used in human medicine as well as cholestyramine, which inhibits enterohepatic circulation of thyroid hormones by binding to the gastrointestinal tract [40–42]. These methods are rarely used in human patients and probably have limited use in veterinary patients with thyroid storm.

Systemic Support

The third arm of treatment for thyroid storm involves reversing the effects of thyroid hormones on the body. Fever should be treated by the judicious use of ice packs and fans. Volume depletion is another common systemic effect of thyroid storm, and this should be treated aggressively with crystalloid fluid replacement. Because many cats have concurrent cardiomyopathy, they should be thoroughly evaluated for heart failure to ensure judicious fluid use. Colloid therapy is generally not indicated unless severe gastrointestinal disease or another syndrome resulting in low oncotic pressure is present. Serum potassium levels should be closely monitored, and potassium supplementation should be added as necessary, remembering that some patients with thyroid storm become acutely hypokalemic and demonstrate severe muscle weakness [25]. Dextrose supplementation of 5% to 10% should be considered as well as B vitamin supplementation to combat potential thiamine deficiency in hyperthyroid cats.

Cardiac disturbances are common with thyroid storm in people, and it is not uncommon for cats with thyroid storm to be presented with cardiac failure that must be managed. β-adrenergic blockade therapy, as described previously, may also be helpful to manage mild cardiac failure because of its effects in reducing the elevated heart rate caused by thyrotoxicosis; however, its use should be avoided in cats presenting with severe heart failure because it could cause lowering of the cardiac output to a dangerous level. Furosemide (1–4 mg/kg administered intravenously or intramuscularly as a bolus when needed, 0.5–2 mg/kg administered by mouth every day), angiotensin-converting enzyme (ACE) inhibitors (enalapril or benazepril at a dose of 0.5–2 mg/kg administered by mouth twice daily), isosorbide dinitrate (0.5–2 mg/kg administered by mouth every 8–12 hours), nitroglycerin (0.5–1.5 administered cutaneously

every 8–12 hours), or hydralazine (0.5–1 mg/kg administered intravenously as a bolus as needed, 0.5–2 mg/kg administered by mouth every 12 hours) may be useful to manage feline heart failure but must be used with care in patients with renal compromise. In all cases, medications should be started at the lowest levels and titrated up to effect and blood pressure must be carefully monitored. Supraventricular arrhythmias are also common in human thyroid storm, with the most common disturbance being atrial fibrillation. In feline patients with thyroid storm, atrial fibrillation can also occur. β-adrenergic receptor blockade, as described previously, is a first-line defense in treating these arrhythmias. Thromboembolic disease may be a sequela in thyrotoxic feline patients, especially those with heart failure or atrial fibrillation [43]. Anticoagulation therapy should be considered to include low-dose aspirin (5 mg per cat every 72 hours), heparin (200–400 U/kg administered subcutaneously every hour until the partial thromboplastin time [PTT] is 1.5–2 times prolonged), and low-molecular-weight heparin (100 U/kg administered subcutaneously every 6 hours). Hypertension is often a complication of thyroid storm in cats. Blood pressure in these cats should be checked, and antihypertensive therapy should be instituted as appropriate to include beta-blockade, as discussed previously, or amlodipine (0.625–1.25 mg per cat every day). In acute cases of hypertension, nitroprusside may be used as a constant rate infusion at 0.5 to 5 μg/kg/min.

In human beings with thyroid storm, a relative adrenal insufficiency can be found because of increased cortisol clearance, leading some physicians to treat with glucocorticoids [42,44]. No such studies have been done in feline patients with thyroid storm, and the use of glucocorticoid therapy in these patients is controversial.

Eradication of the Precipitating Factor

In human thyroid storm, a precipitating factor is one of the criteria that define the disease. The presence of a precipitating factor should be thoroughly investigated in cats presenting with thyroid storm. A full workup, including a full hematologic examination, biochemical analysis, urinalysis, retroviral testing, blood pressure measurement, and imaging studies, should be performed on these cats. Abnormal findings should be further examined by specialized testing. If another abnormality is identified, it should be treated to prevent recurrence of thyroid storm.

OUTCOME

Although thyroid storm is an uncommon presentation in human emergency rooms, there is a significant rate of mortality in patients with this syndrome. Rapid recognition of the problem as well as aggressive treatment is necessary for a successful outcome. Thyroid storm is not as well defined a syndrome in feline medicine, although acute manifestations of thyrotoxicosis result in a syndrome that can be considered feline thyroid storm. Veterinary recognition of this syndrome may be lacking; thus, it is unknown what the true incidence and mortality from thyroid storm may be in cats. Nevertheless, it is certainly recognized that death

may result from treated or untreated acute thyrotoxicosis. As in human patients, it is anticipated that early recognition and aggressive treatment of feline thyroid storm should improve the survival of veterinary patients.

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