Adrenal Disorders in Cats

Pituitary-dependent hyperadrenocorticism is more common in middle-aged to older cats (age range, 5 to 16 years; mean, 10 years) with female cats affected slightly more often. The most common presenting signs are insulin-resistant diabetes mellitus, cutaneous atrophy, polydipsia, polyuria, polyphagia, lethargy, abdominal enlargement, panting, obesity, muscle weakness, and recurrent upper respiratory and urinary tract infections. In some cases the first clinical sign noted may be skin fragility. Diagnosis can be challenging. Unlike in dogs, urine specific gravity is often concentrated despite polyuria/polydipsia, and serum alkaline phosphatase levels are not elevated. High serum alanine aminotransferase levels, hypercholesterolemia, hyperglycemia, and low blood urea nitrogen levels are common. The urinary cortisol/creatinine ratio is a simple and valuable screening tool in cats. The low-dose (0.1 mg/kg) dexamethasone suppression test is the test of choice. Cats do not respond well to mitotane but do appear to respond to trilostane (30–60 mg/cat/day). Therapy is monitored with weekly ACTH stimulation tests. Surgical treatment is an option, but postoperative management is challenging.

Feline hyperaldosteronism, caused by a unilateral aldosterone-secreting adrenal tumor or bilateral hyperplasia, produces oversecretion of aldosterone, which causes hypokalemia, hypernatremia, and metabolic alkalosis. The clinical signs result from associated systemic hypertension. The disease tends to occur in older cats (mean age 10 years). The most common clinical sign in 13 reported cases was hypokalemic polymyopathy presenting as ventriflexion of the neck. Other signs included paresis, hindlimb weakness, hypertension, fundic changes, blindness, polyuria/polydipsia, and polyphagia. Abdominal ultrasonography revealed an enlarged adrenal mass in 11 of 13 cats. Medical treatment of cats with primary hyperaldosteronism consisted of potassium supplementation and aldosterone-blockers such as spironolactone and amiodipine.

COMMENTARY: When feline hyperadrenocorticism was “first” reported, most cats had skin fragility syndrome. I have seen several cats in which this change reversed after treatment. Now that the disease is more commonly recognized, cats are being identified before this dramatic change occurs.—Karen A. Moriello, DVM, Diplomate ACVD


L-Asparaginase in Feline Lymphoma

To evaluate its efficacy as a sole treatment, cats with confirmed diagnosis of lymphoma with no prior history of administration of chemotherapeutic drugs were administered a single IM injection of L-asparaginase (400 IU/kg body weight). Plasma amino acid profiles and ammonia concentrations were determined before, 48 hours after, and 1 week after drug administration to assess changes in plasma concentrations of asparagine, aspartic acid, glutamine, glutamic acid, and overall tumor burden. Results indicated that L-asparaginase significantly reduced asparagine concentrations within 2 days of treatment, although this effect was lost at 1 week after treatment. In the 13 cats in the study, 2 had complete responses (100% regression of measurable lesions) and 2 had partial responses (incomplete but 50% or greater disease regression). Four cats improved clinically but had stable disease; disease progressed in 5 cats, with 3 requiring additional antineoplastic therapy because of rapidly worsening clinical condition during the 7-day study period. The low response rate to L-asparaginase (30%) in this study could be due to inherent asparagine synthetase activity in feline lymphoma cells; inadequate dosing of the drug; and the cat’s innate ability to continuously mobilize and transaminate amino acids, leading to prompt replenishment of asparagine stores.

COMMENTARY: Normal mammalian cells require L-asparaginase to remain viable. Asparagine synthetase maintains adequate levels of L-asparaginase in normal cells. Cells that lack asparagine synthetase, such as those found in lymphoreticular neoplasms, would die if L-asparaginase were depleted. A bacteria-derived enzyme called L-asparaginase has the ability to break down asparagine, which at high enough doses could deplete abnormal cells of asparagine and cause cell death. Although the efficacy of a single agent has not been evaluated extensively, L-asparaginase has been used in multimodal protocols to treat canine and feline lymphoma. The data summarized in this article demonstrate L-asparaginase caused a reduction in plasma asparagine concentrations in cats at the current recommended dose, but the effect was not sustained. Adjustment of the currently recommended dose and dosing interval may be required to improve efficacy of this drug as a chemotherapeutic agent in cats with lymphoma. —The Editors