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FELINE ADRENAL TUMOURS

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Adrenal tumours are an uncommon finding in cats. Based on available data, it is estimated that approximately 0.03% of the feline population (representing 0.2% of all cat tumours) develop a primary adrenal gland tumour. Metastasis to the adrenal glands from other organs is uncommon but when it does occur, lymphoma seems to be the most common (1).
An adrenal tumour may be functional (i.e., producing and secreting a hormone) or nonfunctional. In cats, adrenocortical tumours can secrete excessive amounts of cortisol, progesterone and other sex steroid hormones, or aldosterone. Feline adrenal medullary tumours (phaeochromocytoma), although extremely rare, secrete excessive amounts of catecholamines.

CORTISOL-SECRETING ADRENAL TUMOURS

A cortisol-secreting adrenal mass causing hyperadrenocorticism is the most common functional adrenal tumour identified in cats. Naturally occurring hyperadrenocorticism (Cushing's syndrome) is rare in cats (2-4). Pituitary-dependent hyperadrenocorticism accounts for the majority of cases, but cortisol-secreting adrenocortical neoplasia is responsible in approximately 20% of cats. About one-third of theses adrenal tumours in cats are malignant.
Historical and clinical findings in cats with cortisol-secreting adrenal tumours may include lethargy, weakness, pendulous abdomen, thin fragile skin, bilaterally symmetric alopecia, dull haircoat, seborrhea sicca, muscle atrophy, polyuria, polydipsia, and polyphagia (2-4). In contrast to dogs with hyperadrenocorticism, polyuria and polydipsia in affected cats appear to be secondary to concurrent diabetes mellitus in the vast majority of cases. Hyperglycemia and glycosuria are seen in up to 90% of cats and hypercholesterolemia and elevated serum ALT activity are common. However, a high serum alkaline phosphatase activity is not a consistent finding in cats with hyperadrenocorticism.
In addition to the typical clinical signs and clinicopathologic findings associated with hyperadrenocorticism, diagnosis of hyperadrenocorticism due to a functional adrenal tumour is confirmed using tests of pituitary-adrenocortical axis (ie, high dose dexamethasone suppression test and endogenous ACTH concentrations). Finally, imaging studies (ie, abdominal ultrasound, CT, MR) should be used to confirm the presence of an adrenal tumour in these cats (2-4). In cats with adrenal-dependent hyperadrenocorticism, the contralateral adrenal gland is expected to be small or atrophied as a result of suppressed pituitary ACTH secretion.
Unilateral adrenalectomy is most successful method of treating cats with cortisol-secreting adrenocortical tumour (2-5). In cats with adrenal adenoma or adrenal carcinoma that has not yet metastasized, adrenalectomy may be curative. If tumour resection is successful, circulating cortisol fall to low concentrations and these cats generally require glucocorticoid supplementation for approximately two months postoperatively until the glucocorticoid secretory function of the atrophied contralateral gland recovers.
Because of the deleterious effects of chronic cortisol excess on skin fragility as well as on immune and cardiovascular function, many cats with untreated hyperadrenocorticism are poor surgical candidates. Surgery has been difficult to perform owing to the debilitated condition of these cats. Although further investigation needs to be done, trilostane (5-15 mg/kg) appears to be useful in the preoperative preparation of these cats prior to adrenalectomy (3,6). In those cats that are not surgical candidate or have adrenal tumour metastasis, trilostane may also be useful in their long-term management, at least for a few weeks to months.

SEX HORMONE SECRETING ADRENAL TUMOURS

A functional tumour arising from the adrenal cortex could secrete excessive amounts of adrenal progestagens, androgens, or estrogens. Progesterone-secreting adrenal tumours have been the most common sex hormone secreting adrenal tumour reported in cats (6-10). Clinical signs are

similar to those in cats with cortisol-secreting tumours. Excessive progesterone secretion in affected cats causes diabetes mellitus and feline fragile skin syndrome, which is characterized by progressively worsening dermal and epidermal atrophy, endocrine alopecia, and easily torn skin. In most of these cats with progesterone-secreting adrenal tumours, results of tests of the pituitary-adrenocortical axis are normal to suppressed and the contralateral adrenal gland is normal in size and shape on abdominal ultrasound. Diagnosis requires documenting an increased concentration of one or more adrenal sex steroids, ideally measured before and after ACTH stimulation.
Recently, a male cat that had developed strong urine order and aggressive behaviour was documented to have a functional adrenal adenoma associated with high circulating concentration of androstenedione and testosterone (11). After adrenalectomy, serum concentrations of the androgens decreased and urine spraying urine aggression resolved.

ALDOSTERONE SECRETING ADRENAL TUMOURS
Primary hyperaldosteronism (Conn's syndrome) appears to be a relatively rare but greatly underdiagnosed disease of older cats. This syndrome is characterized by excessive autonomous secretion of aldosterone from one or both adrenal glands, resulting in clinical signs relating to hypertension and/or hypokalaemia (13-16).
About half of cases have been due to unilateral aldosterone-secreting adrenal adenomas, whereas most of the remaining cats have unilateral adrenal carcinomas. Less commonly, bilateral adrenal adenomas or bilateral adrenal hyperplasia (17) have been reported. Occasionally, an aldosterone-secreting adrenal tumour is also found to be hypersecreting another adrenocortical hormone, most commonly progesterone (9,11); these cats also had diabetes mellitus and dermatologic changes, both attributed to progesterone excess rather than hyperaldosteronism.
Aldosterone is the major mineralocorticoid secreted by the adrenal cortex and is responsible for regulation of sodium and potassium balance. Therefore, the hormone helps maintain intravascular fluid volume and acid-base balance. Historical findings are generally nonspecific can include generalized weakness (sometimes episodic), lethargy, stiffness, muscle pain, polyuria/polydipsia, and blindness (13-17). Physical examination findings might include ventroflexion of the neck, hypertension, blindness, and retinal vessel tortuosity.
Cats with hyperaldosteronism commonly have moderate to severe hypokalaemia and metabolic alkalosis. The sodium concentration is normal to mildly elevated. Demonstration of an inappropriately elevated serum aldosterone concentration along with a low plasma renin concentration provides a definitive diagnosis of hyperaldosteronism. Ideally a diagnosis is made on the basis of marked hyperaldosteronemia in conjunction with hypertension, hypokalaemia, inappropriate kaliuresis (high urinary fractional excretion of potassium), and low plasma renin activity (14-17). The presence of renal failure presents a particular diagnostic dilemma, as renal failure itself can lead to a similar constellation of abnormalities. The magnitude of aldosterone elevation may be the key (ie, aldosterone is only about 2-3 times normal with renal failure).
A recent report assessed changes of the urinary aldosterone-to-creatinine ratio in normal cats in response to increased dietary salt or administration of fludrocortisone acetate (18). In that study, normal cats showed the most consistent decrease of the urinary aldosterone-to-creatinine ratio with administration of fludrocortisone acetate as compared with dietary salt supplementation. One cat with an aldosterone-secreting adrenal carcinoma had an elevated ratio and no suppression in response to fludrocortisone acetate. Such mineralocorticoids function tests may prove useful as more cats are diagnosed with this syndrome.
Initial treatment of cats with hyperaldosteronism should be directed toward provision of parenteral or oral potassium supplementation and correction of any fluid deficits and acid-base imbalances. For this purpose, potassium gluconate is generally given at the dosage of 2-6 mEq/day, with the dose adjusted as necessary to maintain normokalaemia. If necessary, the diuretic spironolactone, which acts as an aldosterone receptor antagonist, can also be administered at the dosage of 2-4 mg/kg/day.
Surgical adrenalectomy is the treatment of choice in most cats with hyperaldosteronism that do not have evidence of metastatic disease. For those cats that have bilateral adrenal hyperplasia, metastatic disease, or whose owners have declined surgery, medical management with oral spironolactone and potassium can be continued indefinitely.

CATECHOLAMINE SECRETING ADRENAL TUMOURS

Phaeochromocytoma is a catecholamine-producing tumour derived from the chromaffin cells of the adrenal medulla that is extremely rare in cats (1, 2). Clinical signs and physical examination findings develop as a result of the space-occupying nature of the tumour and its metastases, or as a result of excessive secretion of catecholamines and their impact on blood pressure and cardiac function. A diagnosis of phaeochromocytoma prior to surgery is usually one of exclusion. Unlike a cortisol-secreting adrenal tumour, the contralateral adrenal gland should be normal in size and shape with a catecholamine-producing adrenal tumour. Catecholamine secretion by the tumour, and thus systemic hypertension, tends to be episodic; failure to document systemic hypertension does not rule out phaeochromocytoma. Measurement of urinary catecholamine concentrations or their metabolites can strengthen the tentative diagnosis of phaeochromocytoma but is not commonly performed in cats. Because many of the clinical signs and blood pressure alterations are similar for phaeochromocytoma and adrenal-dependent hyperadrenocorticism, it is important to rule out adrenal-dependent hyperadrenocorticism before focusing on phaeochromocytoma.

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