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Opinion Article

Progression of Adrenocortical Tumors in Animals

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DESCRIPTION

The surgical treatment of adrenal tumors has evolved over the last century, as our understanding of what hormones the adrenal glands secrete and what those hormones do. This article provides an overview of the preoperative evaluation of patients with adrenal tumors, which can be benign or malignant, including metastases. The biochemical assessment of excessive hormone levels is discussed, as are imaging features that distinguish benign from malignant tumors. Options for the surgical management are described, including the advantages and disadvantages of various open and laparoscopic approaches. The surgical management of adrenocortical carcinoma is specifically reviewed, including controversies surrounding surgical approaches, as well as the surgical management of invasive or recurrent disease. Hyper adrenocorticism is defined as the excessive production of steroid hormones by the adrenal gland.

The most common cause of hyper adrenocorticism in cats is a functional pituitary tumor that secretes Adrenocorticotropic Hormone and Corticotropin (ACTH). However, cortisol-secreting adrenal tumors are rare in cats and functional cortisol-secreting adrenal tumor can also cause hyper adrenocorticism. Adrenal cortical tumors can also secrete other hormones, including mineralocorticoids and adrenal sex hormones. Adrenal adenomas and adenocarcinomas are rare in cats and represent 0.2% of all cancers of this type. Most primary adrenal tumors in cats secrete aldosterone, although there have been some reports of secretion of the sex hormone cortisol and co-secretion of cortisol-progesterone, cortisol-aldosterone, aldosterone-progesterone and cortisol- has documented progesterone or testosterone in cats.

This study describes an unusual case of a cat diagnosed with an adrenal cortical tumor secreting more than one type of steroid hormone and successfully treated with a unilateral adrenalectomy. Cats had clinical signs consistent with both hyper adrenocortisim and hyperthyroidism. Unfortunately, circulating thyroxine levels were not measured prior to adrenalectomy. However, the complete disappearance of clinical signs after adrenalectomy argues against hyperthyroidism as the cause of the clinical signs before adrenalectomy. Unlike dogs, cat renal tubules are not very sensitive to cortisol. Consequently, hyper cortisolism does not automatically lead to polyuria and polydipsia in this species. However, most, but not all, cats with hyper cortisolism develop secondary diabetes mellitus resulting in polyuria, polydipsia and polyphagia.

The increase in cortisol levels observed after ACTH administration indicated hyper cortisolism. Left adrenal hypotrophy may also be consistent with a cortisol-secreting adrenal cortical tumor, resulting in low pituitary secretion of ACTH. Hyper cortisolism due to an adrenocortical tumor could not be clearly demonstrated because the endogenous ACTH concentration was not measured. In the presence of an adrenocortical tumor, clinical signs of hyper adrenocorticism may occur even when basal cortisol levels are low. Aldosterone is the main mineralocorticoid hormone produced by the zona glomerulosa of the adrenal cortex. The main function of aldosterone is to regulate blood pressure by increasing water, sodium reabsorption and potassium secretion in the distal renal tubules, colon and salivary glands.

In primary hyper aldosteronism, aldosterone is secreted autonomously by the adrenal cortex, leading to sodium retention and systemic hypertension. Primary hyper aldosteronism can be caused by unilateral or bilateral nodular hyperplasia, adenomas or adenocarcinomas of the zona glomerulosa. Secondary hyper aldosteronism can occur in chronic heart or kidney disease due to over activity of the renin-angiotensin system. Secondary hyper aldosteronism was excluded on the basis of normal cardiac and renal findings. The suspicion of primary hyper aldosteronism was based on the finding of increased serum aldosterone concentration before treatment and decreased aldosterone concentration after surgical removal of the adrenal tumor, but plasma renin activity was not measured. Cats with adrenal cortical tumors can also hyper secrete sex steroids. In this study, a high serum concentration of progesterone was observed in the cat.

Studies have shown that high serum progesterone levels can increase free cortisol levels by displacing cortisol from the cortisol-

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binding protein, leading to hyper cortisolism. In addition, high adrenal progesterone production can cause hyper secretion of mineralocorticoids, glucocorticoids and androgens. Since progesterone synthesized in the adrenal cortex can be converted into these hormones. Interestingly, adrenal tumors that secrete high levels of serum progesterone can cause mineralocorticoid excess syndrome even in the absence of hyper aldosteronism. A high serum concentration of progesterone was observed in the cat discussed here, which could also explain the high serum concentration of aldosterone.

In general, adrenalectomy is the treatment of choice for a non-metastatic unilateral functional adrenal tumor. In cats, after complete removal of a functional adrenal tumor, hypokalaemia and hypertension resolve in the immediate postoperative period and no further drug therapy is required. With medical treatment alone, the prognosis may not be favorable as cats become resistant to potassium supplementation and antihypertensive drugs. However, it has been reported that perioperative bleeding is an important and common complication in cats with aldosterone-secreting adrenal tumors.