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Claudia E. Reusch
Prof. Dr., Dipl ECVIM-CA
Clinic for Small Animal Internal Medicine, University of Zurich, Switzerland

The first case of feline primary aldosteronism was described in 1983. Since then, the disease has been diagnosed with increased frequency. Although no data are available concerning the true prevalence, it is assumed that the disease is more common than initially thought. This hypothesis is based on data from human medicine, where increased disease awareness led to a more systematic screening of the hypertensive population resulting in a strong increase in prevalence. Primary aldosteronism however, seems to be very rare in dogs.
In human medicine, approximately two-thirds of patients have bilateral idiopathic adrenal hyperplasia while about one third has aldosterone-producing adenomas (aldosteronomas). Typical findings are systemic hypertension, hypokalemia and metabolic acidosis. However, because screening for the disease is becoming more systematic and the diagnosis is generally made earlier, the prevalence of hypokalemia is decreasing and nowadays the majority of patients are normokalemic at the time of diagnosis. Currently, 5 to 10% of the general hypertensive population and 20% of patients with severe or resistant hypertension are assumed to suffer from primary aldosteronism. The degree of hypertension is usually moderate to severe, and patients with aldosteronoma tend to have higher blood pressure than patients with idiopathic aldosteronism.
The consequences of increased aldosterone concentration are retention of sodium and water in the distal and collecting tubules of the kidneys. This results in increased intravascular volume and increased urinary potassium and hydrogen excretion. Excessive concentrations of circulating aldosterone also induce vasoconstriction and lead to an increase in peripheral vascular resistance. The two central mechanisms responsible for the development of hypertension in primary aldosteronism are expansion of plasma and extracellular fluid volume and increase in total peripheral vascular resistance. Aldosterone *per se* has pro-inflammatory and pro-fibrotic properties resulting in vascular, cardiac and renal lesions. The pathophysiology of aldosterone-associated hypertension in cats is thought to be identical to humans.
The majority of cats with primary aldosteronism have been shown to have unilateral carcinomas, while adenomas and hyperplasia have been less frequently reported. Clinical signs include weakness with associated cervical ventroflexion, mydriasis and blindness because of hypertensive retinopathy; some cats also show polyuria/polydipsia.
Almost all cats described to date have been hypokalemic at the time of diagnosis. However, as in human medicine, it may be possible that aldosteronism is overlooked in cats with normal potassium levels. A more systematic screening for primary aldosteronism may improve diagnosis and thus increase the prevalence of the disease. Based on data available to date, the prevalence of hypertension in cats with primary aldosteronism appears to be high. Blood pressure was recorded in 30 cases, 26 of which were hypertensive. The severity ranged from mild to severe (185-270 mmHg), and the most common sequels were retinal detachment and ocular bleeding. Definitive diagnosis requires demonstration of inappropriately elevated aldosterone concentration with low plasma rennin activity. The disease is often suspected only after the finding of an adrenal mass by ultrasonography.
Initial treatment should be directed towards alleviation of hypertension and hypokalemia by using an aldosterone antagonist (spironolactone 2.5 mg/kg q24h or 6.25 mg/cat q12h PO) and a calcium channel blocker (amlodipine besylate 0.625-1.25 mg/cat q24h PO), and substituting potassium as needed. Subsequent adrenalectomy is the treatment of choice for animals

without tumor metastasis. In the few cases described in the literature, as well as in the cases seen at our hospital, hypertension resolved after surgery. In cases in which adrenalectomy is not feasible (e.g. metastasized tumor, bilateral tumor or hyperplasia), medical treatment with spironolactone and amlodipine besylate should be continued. The two drugs combined seem to lead to resolution of hypertension in most cases.

References

Ash RA, Harvey AM, Tasker S. Primary hyperaldosteronism in the cat: a series of 13 cases. J Feline Med Surg, 2005;7:173-182

Reusch CE, Schellenberg S, Wenger M: Endocrine hypertension in small animals. Vet Clin North Am Small Anim Pract, 2010;40:335-352

Willi B, Kook PH, Quante S, Boretti F, Sieber-Ruckstuhl NS, Grest P, Scherrer O, Riond B, Hofmann-Lehmann R, Nussberger J, Reusch CE: Primary hyperaldosteronism in cats. Schweiz Arch Tierheilk, 2012;154:529-537