Pacific Tide

An informational newsletter

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About our Author

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Dr. Akol graduated from UC Davis in 1987 with a Doctorate in Veterinary Medicine. Her internship at Coast Pet Clinic in Hermosa Beach followed and was completed in 1988. Dr. Akol then pursued residency training in internal medicine at University of Pennsylvania (1988-1990). She became board certified in internal medicine in 1992. She has published articles in the area of diabetes mellitus, pancreatitis and lipidosis. Dr. Akol has a special interest in feline medicine as well as the sub-specialties of gastroenterology, endocrinology and diseases of the respiratory system.

Dr. Akol enjoys in her free time competing in ballroom dancing and has won titles nationally. She enjoys watching her son play soccer and spending time with her two dogs, Keeper and Pink.

Kelly Akol, DVM, DACVIM

Feline Hyperaldosteronism

By: Kelly Akol, DVM, DACVIM (SAIM)

Primary hyperaldosteronism (PHA) is caused by adrenocortical autonomous secretion of aldosterone. This disease was first discovered in humans in 1953. Initially considered rare, is it now estimated to account for 5-13% of all people with arterial hypertension. To date, 49 cases of feline PHA have been reported in the literature. Like humans, it is currently very likely to be underdiagnosed. The inappropriately high aldosterone secretion can come from unilateral or bilateral neoplasia, or bilateral nodular hyperplasia of the adrenal zona glomerulosa. Awareness of the pathophysiology of the disorder can lead to a heightened index of suspicion in older patients with hypertension and/or hypokalemia, and successful therapeutic intervention.

Aldosterone regulates systemic blood pressure and extracellular fluid volume in response to changes in renal blood flow and electrolytes. Under normal conditions, if the kidneys experience decreased blood flow, the renin-angiotensin-aldosterone-system is activated. This causes increased sodium retention, increased extracellular fluid volume and renal potassium excretion. In PHA, excess production of aldosterone causes hypertension and profound hypokalemia without an increase in renin. Aldosterone also has proinflammatory and profibrotic properties that may promote or accelerate progressive renal disease.

The combination of elderly feline patients with chronic kidney disease, low potassium and hypertension is a common clinical finding, but only a fraction of them have PHA. Primary diseases in geriatric felines that cause reduced renal blood flow such as heart disease, hypoproteinemia and chronic kidney disease can all lead to renin dependent secondary hyperaldosteronism. Hypokalemic nephropathy is a subset of patients with secondary hyperaldosteronism that have developed hypokalemia. PHA patients are usually elderly, may have chronic kidney disease and have hypokalemia and/or hypertension, but the aldosterone production is renin independent production by the zona glomerulosa of the adrenal gland(s) due to neoplasia or hyperplasia. How do you distinguish this subset?

Forty-nine cases of presumed or confirmed feline PHA have been reported. Median age was 13 years, with a range of 5-20 years. There is no sex or breed predilection. Physical examination findings were related to renal disease, hypertension and potassium depletion. Ocular manifestations of hypertension may include mydriasis, hyphema, and blindness due to retinal detachment and/or ocular hemorrhage. Muscle weakness is the hallmark of hypokalemia, but there is not a strict correlation with severity. Signs can include acute, generalized weakness, plantigrade stance, cervical ventroflexion, inability to jump and lateral recumbancy. 43/49 cases had hypokalemia, 19/37 had azotemia, and 37/49 has hypertension. Cats with PHA tend to have low or low normal phosphorus compared to cats with secondary hyperaldosteronism. Plasma aldosterone concentration was increased in 39/49; plasma renin was below or within normal limits in the 22 cats in which it was measured.

Plasma aldosterone concentration test is a good screening test, but is not elevated patients in all patients. In the presence of hypokalemia, even a mildly elevated aldosterone level is inappropriate. The gold standard screening test is the aldosterone-to-renin ratio. Aldosterone can be readily measured, but renin meas-

urement has some very challenging practical obstacles. It must be cooled immediately and shipped overnight on dry ice. Very few labs are able to run this test, and none have a reference range. Another appropriate test is the urine-aldosterone-to-creatinine ratio. This can reflect aldosterone values over a little longer time interval than the plasma level. However, this test can also be normal in some patients. The sensitivity of the test can be improved by running the test before and after a four day treatment with fludrocortisone – called the fludrocortisone suppression test. Unfortunately, the aldosterone-to-renin ratio and the urine-aldosterone-to-creatinine ratio can both be influenced by concurrent medication such as diuretics and antihypertensives. Many of these patients would decompensate if these medications were discontinued for the suggested two week period prior to testing.

In suspect patients, adrenal gland imaging can be done with ultrasound, CT or MRI. In cases of unilateral neoplasia without evidence of metastasis, surgery can be curative. Patients with bilateral hyperplasia, or patients for whom surgery is not an option, can benefit from medical management. Care must be taken in interpretation of these imaging studies. In 5 of 30 cats with histologically confirmed PHA, the adrenal diagnostic imaging results were inaccurate, leading to inappropriate therapy.

Unilateral adrenalectomy is the treatment of choice for patients with confirmed unilateral PHA; however, surgery is associated with complications such as abdominal hemorrhage. Perioperative complications occurred in 8 of 24 surgical cases reported. Six of these patients died. For those with hyperplasia, milder neoplasia, or poor surgical candidates due to metastasis or other complications, medical management with spironolactone and oral potassium supplementation to control the hypokalemia, and amlodipine to treat hypertension can be very successful.

Case Report:

Signalment-6 year old MN DMH.

Past history- He had been a fairly stable Diabetic for the previous 3 years. He had developed subclinical, mild hypokalemia 9 months prior and had been well-controlled with oral potassium supplementation.

The cat presented for lethargy. Appetite was stable. He had an intermittent 3 beat gallop and stable weight. He was lethargic and a little weak. Potassium was below normal at 2.4 mmol/L (2.9-4.2). His potassium dose was increased by 30%. Recheck one week later revealed more dramatic gallop rhythm and further reduction in potassium to 2.1 mmol/L. Potassium dose was increased by 50%. This amount of potassium was causing him to vomit and he lost 13 ounces in the ensuing week; he was unable to blink the left eye. Potassium level dropped further to 2.0 mmol/L. BUN and creatinine were normal at 27 mg/dL and 1.5 mg/dL respectively. Phosphorus was normal at 3.3 mg/dL. Magnesium level was normal, as were sodium and chloride. Systolic blood pressure on Doppler was mildly elevated at 170 mmHg. Plasma aldosterone was 3482 pmol/L (194-388). Abdominal ultrasound revealed a 2.6x2.0 cm mass on the left adrenal gland. The mass had an irregular contour and invasion into adjacent fat. A diagnosis of invasive, adrenal tumor causing autonomous aldosterone secretion was made. The patient was not a candidate for adrenalectomy. The formulation of his potassium supplementation was changed to be better tolerated and he started on 2mg/kg spironolactone BID. Recheck in two weeks brought normalization of his blink, resolution of the gallop rhythm and lethargy, and normalization of potassium to 3.1 mmol/L.

Our Doctors

Internal Medicine
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Merrianne Burtch, DVM, DACVIM(SAIM)
Michelle Pressel, DVM, DACVIM (SAIM)
Bryn Hoffman, MVB (Residency Trained in Internal Medicine)

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Tom LaHue, DVM, DACVS

Critical Care
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Cardiology
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Larry Kerr, DVM, DACVR
Mark Lee, DVM, DACVR

Emergency
Christian Robison, DVM
Mark Saphir, DVM
Jessica Kurek, DVM
Sara Heidelberger, DVM

Behavior
Jan Brennan, DVM (practice limited to behavior)

About Our Hospital

Pacific Veterinary Specialists was founded to provide high quality, specialized medical care to companion animal patients. Our practice is dedicated to serving the veterinary community as a partner in total patient care. We offer comprehensive specialized services including video endoscopy, Doppler ultrasound, surgery, 24-hour ICU care, and emergency and critical care. Our staff is committed to providing compassionate and thorough medical care that meets the needs of the patient, client, and referring veterinarian.

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