

Feline Hyperaldosteronism

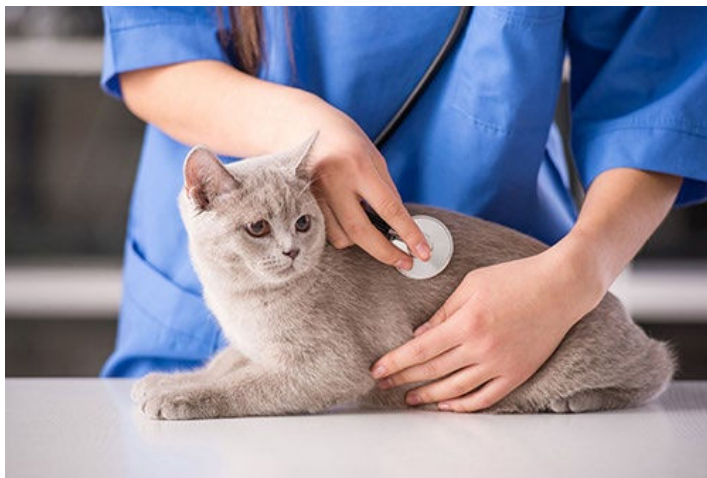


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Aldosterone Physiology

The zona glomerulosa is the layer of the adrenal gland that secretes aldosterone, the principal mineralocorticoid responsible for regulating sodium and potassium. An increase in potassium directly stimulates aldosterone secretion from the adrenal glands. In addition to a decrease in potassium, a decrease in blood pressure will stimulate the renin-angiotensin-aldosterone system (RAAS), leading to increased sodium resorption and potassium and hydrogen excretion. The sodium resorption leads to the conservation of water, raising blood volume. The RAAS system will also increase systemic vascular resistance due to angiotensin II. The vast majority of physiologic actions in the RAAS system are mediated by angiotensin II. In addition to regulating vascular resistance, angiotensin II contributes to cell growth, aldosterone production, and controlling the glomerular filtration rate and renal blood flow.

Mineralocorticoids have two essential roles: they regulate extracellular fluid volume and are the major regulator of potassium homeostasis. The kidney is the primary target tissue for mineralocorticoids, the distal convoluted tubule specifically. They bind to receptors in the epithelial cells, activating sodium channels and stimulating the Na^+ , K^+ -ATPase. An electrochemical gradient is established, leading to passive excretion of potassium into the urine.



Feline Hyperaldosteronism

Hyperaldosteronism can be classified as primary (PHA) or secondary. Primary hyperaldosteronism occurs when adrenocortical cell secretion of aldosterone is autonomous and does not respond to negative feedback, i.e., an adrenocortical tumor. The tumor is usually unilateral and can have varying degrees of malignancy, ranging from well-capsulated adenomas to carcinomas with caudal vena cava invasion. Secondary hyperaldosteronism occurs when some other disease process, such as congestive heart failure renal panel, stimulates RAAS activation, leading to aldosterone production.

The incidence and recognition of feline PHA have been increasing over the past 20 years. There is no breed predisposition. The median age at diagnosis is around 13 years old; the majority are over 10. There is no sex predisposition. Clinical signs are primarily related to hypokalemia (hindlimb weakness, ventroflexion, ataxia, etc.) and hypertension (sudden blindness, retinal detachment, seizures). Weakness is usually apparent on physical examination.

Diagnosis



Cervical ventroflexion from hypokalemic myopathy

Routine laboratory testing reveals hypokalemia. An increased urinary fraction of potassium can confirm that the hypokalemia is renal in origin, but this is rarely performed. Hypernatremia is uncommon due to the “Aldosterone escape” phenomenon, which is volume or pressure natriuresis. Creatine Kinase levels are variably elevated with hypokalemic myopathy. Imaging is of variable helpfulness. A large tumor may be detected on ultrasound unilaterally with a small contralateral adrenal gland. Bilateral adrenal tumors and pulmonary metastasis are both rare. Abdominal CT and MRI may be helpful, but the number of reports is limited. Confirmation of hyperaldosteronism is obtained by measuring a high basal aldosterone in the face of hypokalemia. This is not a useful test to determine whether the hyperaldosteronism is primary and secondary, as results overlap. Plasma renin activity could be used to determine if

it is primary (decreased or normal due to negative feedback) or secondary (increased due to RAAS activation), but this test is not available in the United States.

Treatment and Prognosis

The treatment of choice for a feline aldosteronoma or other adrenal tumors is an adrenalectomy. This is potentially curative but requires highly skilled surgeons and is highly risky. An increase in anesthesia time has been associated with decreased survival. The hypokalemia should be corrected with parenteral or oral potassium. It is also essential to assess for metastasis and tumor thrombi. It is crucial post-operatively to monitor electrolytes as the negative feedback has put the zone glomerulosa cells of the contralateral adrenal gland into dormancy.

Medical management for cats that do not undergo an adrenalectomy focuses on increasing the potassium and regulating the blood pressure. Potassium supplementation (2-6 meq PO q12h) has been effective. Amlodipine (0.625-1.25 mg/cat PO q24h) is the treatment of choice for hypertension. Up to 2.5 mg/cat can be used if the blood pressure is not controlled on the conventional doses. Spironolactone can be used (2-4 mg/kg PO q24h) to decrease blood pressure and increase serum potassium. Medical management has yielded a median survival time of several months to a couple of years.

References

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