Diagnosing Hypoadrenocorticism

Hypoadrenocorticism (HA) occurs when the adrenal gland secretes insufficient amounts of glucocorticoids and, in the case of primary HA, mineralocorticoids; it is usually caused by immune-mediated destruction of the adrenal cortex. Affected dogs are presented with a host of nonspecific signs as well as hyponatremia, hyperkalemia, and a low sodium-to-potassium ratio (SPR). Although highly suggestive of HA, these signs are sometimes absent and are not pathognomonic. The adrenocorticotropic hormone (ACTH) stimulation test is the favored test for definitive HA diagnosis. However, shortages of the synthetic ACTH needed to perform the test and cost increases are prompting a search for alternative testing.

This study prospectively investigated cortisol-to-ACTH ratio (CAR), plasma ACTH, baseline cortisol concentrations pre-ACTH stimulation testing, and SPR as potential screening tests in dogs with confirmed HA ($n = 23$), diseases mimicking HA ($n = 79$), and healthy dogs ($n = 30$). Baseline serum cortisol concentrations, CAR, and SPR were all significantly lower for dogs with HA than for the other groups, whereas plasma ACTH concentrations were significantly higher for the HA dogs. Some overlap was found between the groups, however. The area-under-the-curve calculation suggested that the best option to diagnose HA is the CAR with a sensitivity of 100% and a specificity of 99% when using a cutoff ratio of >0.01. CAR appears to be a promising screening test for primary HA, but the overlap with dogs with non-HA illness should be pursued further, as should the utility of CAR in diagnosing secondary HA.

Global Commentary

Measurement of cortisol concentration, before and after stimulation with a synthetic ACTH preparation, is still my test of choice for diagnosing HA. At present, synthetic cosyntropin (known as tetracosactide in Europe) is supplied by human and (recently) veterinary manufacturers and is widely available, despite shortages in some parts of the world. I advise that practitioners should use cosyntropin and perform ACTH stimulation tests when possible. In absence of synthetic ACTH, I have resorted to measuring basal cortisol and, occasionally, plasma ACTH concentrations. Despite promising results, I believe that the measurement of plasma ACTH concentration and CAR might not become popular because of special sample-handling issues and high cost of the ACTH assay.—*Alice Tamborini, DVM, MRCVS, DECVIM-CA (Internal Medicine)*

Source

Boretti FS, Meyer F, Burkhardt WA, Riond B, Hofmann-Lehmann R, Reusch CE, Sieber-Ruckstuhl NS. Evaluation of the cortisol-to-ACTH ratio in dogs with hypoadrenocorticism, dogs with diseases mimicking hypoadrenocorticism, and in healthy dogs. *JVIM*. 2015;29(5):1335-1341.

Research Note: Plasma & Kidney Disease

Azotemic chronic kidney disease (CKD) is reported in 31% of cats over 15 years of age. Chronic kidney disease–mineral and bone disorders (CKD-MBD) is the syndrome of clinical, biochemical, and imaging abnormalities correlated with CKD and renal osteodystrophy. Hyperparathyroidism has been documented as the most common calcium-phosphate derangement of cats with CKD. The phosphatonin fibroblast growth factor-23 (FGF-23) has been shown to increase with advancing International Renal Interest Society (IRIS) stage of CKD. FGF-23, secreted in response to hyperphosphatemia and increased plasma calcitriol concentrations, acts in the kidney to inhibit calcitriol production and increase phosphaturia. It also decreases parathyroid hormone (PTH) production and secretion in the parathyroid gland. Feline plasma FGF-23 increases prior to the development of overt hyperphosphatemia; therefore, it may better reflect total-body phosphate retention during mild-to-moderate CKD than plasma phosphate concentrations. In human patients, plasma FGF-23 concentrations are an independent predictor of CKD progression.

The objective of this retrospective study was to investigate the relationship between plasma FGF-23 and PTH concentrations at CKD diagnosis with survival time and disease progression over a 12-month period in a group of cats with azotemic CKD. In the final multivariable model, survival was negatively associated with plasma creatinine and FGF-23 concentrations, urine protein-creatinine ratio, and age. It was positively associated with PCV. Independent predictors of disease progression within 12 months included FGF-23 and age. Further research is needed to determine whether FGF-23 is a uremic toxin itself or simply a surrogate marker for other causes of uremic toxicity, but it may serve as a useful biomarker for CKD prognosis.

Source

Geddes RF, Elliott J, Syme HM. Relationship between plasma fibroblast growth factor-23 concentration and survival time in cats with chronic kidney disease. *JVIM*. 2015;29(6):1494-1501.