Atypical Hypoadrenocorticism in Dogs

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In the Literature

Wakayama JA, Furrow E, Merkel LK, Armstrong PJ. A retrospective study of dogs with atypical hypoadrenocorticism: a diagnostic cut-off or continuum? *J Small Anim Pract*. 2017;58(7):365-371.

FROM THE PAGE ...

Atypical hypoadrenocorticism (AH) is an uncommon veterinary endocrinopathy that is classically considered to be an isolated deficiency of cortisol production with normal electrolyte concentrations. Recent evidence suggests that insufficient aldosterone production is frequently present on AH diagnosis, regardless of measured electrolyte concentrations.[1] Because AH can present with various clinical signs or biochemical abnormalities, it is said to mimic many disease states, thereby obscuring consideration of AH as a differential. Confirmation is obtained generally by documenting a post-ACTH stimulated cortisol concentration of less than 2 µg/dL (55 nmol/L). However, suboptimal stimulation (>2 µg/dL [>55 nmol/L] but below the laboratory reference interval) provides equivocal diagnostic information.

This study retrospectively reviewed approximately 10 years' worth of medical records to extract clinical and biochemical data in dogs with confirmed AH (n = 40; stimulated cortisol concentration, 1-1.2 µg/dL [<28-33 nmol/L]) and suspected AH yielding suboptimal ACTH stimulation test results (n = 9; stimulated cortisol concentration, 3.4-8.1 µg/dL [94-223 nmol/L]).

Unlike previous reports in which female dogs were primarily affected, neutered male dogs comprised 57.5% of the AH group, with Labrador retrievers and standard poodles disproportionately affected. Clinical signs of both groups were nonspecific and chronic (present for >3 weeks), with lethargy and GI upset (eg, anorexia, vomiting, diarrhea) observed in most cases.

Hypoalbuminemia and hypocholesterolemia were the most common biochemical abnormalities detected in both groups and were encountered more frequently in confirmed AH dogs as compared with dogs suspected of having AH. Imaging was performed in only a minority of dogs in both groups, and small adrenal gland size was documented only in dogs with confirmed AH. Only 5 of 35 AH dogs developed a low sodium:potassium ratio (≤25.7) within 51 months of diagnosis. Daily physiologic glucocorticoid supplementation resolved clinical signs (30/31), hypoalbuminemia (25/27), and hypocholesterolemia (23/25) in AH dogs. At follow-up for 7 of the 9 dogs with suspected AH, none developed an electrolyte disorder. Several dogs with suspected AH (4/7) were eventually diagnosed with inflammatory bowel disease; clinical signs resolved for 2 of these dogs with no sustained therapy. One dog had persistent signs with no diagnosis obtained.

... TO YOUR PATIENTS

Key pearls to put into practice:

1. AH screening is warranted for patients presented with chronic lethargy or vague GI signs, hypoalbuminemia, and/or hypocholesterolemia.
2. Although electrolyte levels are commonly normal on AH diagnosis, assessment of pre- and post-ACTH stimulated aldosterone concentrations may more accurately reflect the patient's mineralocorticoid status.
3. Suboptimal ACTH stimulation test results may suggest the presence of another occult nonadrenal disease state (eg, enteropathy).

Reference

1. Baumstark ME, Sieber-Ruckstuhl NS, Muller C, Wenger M, Boretti FS, Reusch CE. Evaluation of aldosterone concentrations in dogs with hypoadrenocorticism. *J Vet Intern Med*. 2014;28(1):154-159.