Some Dogs Just Do Not Read the Same Book

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An 8-year-old, castrated male Labrador retriever was presented for anorexia and marked lethargy for the past 2 days.

Diagnostic Testing

Variable	*Result*	*Reference Range*
Total WBC count	34,700/µl	6000–17,000/µl
Segs	24,000 /µl	3000–11,500/µl
Lymphocytes	8000/µl	1000–4800/µl
Monocytes	1400/µl	200–1400/µl
Eosinophils	1400/µl	100–1300/µl
PCV	33%	37%–54%
Platelets	181,000/µl	160,000–430,000/µl
Total protein	4.8 g/dl	5.6–7.4 g/dl
Albumin	1.6 g/dl	2.8–3.8 g/dl
Glucose	51 mg/dl	79–127 mg/dl
Cholesterol	88 mg/dl	139–342 mg/dl
BUN	28 mg/dl	6–27 mg/dl
Na+	145 mEq/L	143–153 mEq/L
K+	4.0 mEq/L	3.5–5.3 mEq/L
Cl	121 mEq/L	107-121 mEq/L
Total CO_2	16 mEq/L	16–27 mEq/L
Urinalysis		
Specific gravity	1.019	
pH	7.5	
Protein	+1	
Glucose	Negative	
Ketones	Negative	
Bilirubin	2+	
Hemoprotein	Negative	
Bacteria	Negative	
Casts and crystals	Negative	
Epithelial cells	0–2/LPF	
RBCs	0–1/HPF	
WBCs	0–1/HPF	

History. The patient, a well-trained, performing search dog, became chronically ill approximately 5 years before. At that time it had been diagnosed and treated for chronic ehrlichiosis and had responded well to several 2- to 4-week cycles of doxycycline treatment. An ACTH-stimulation test was also done at that time because of chronic lethargy. The results showed a resting serum cortisol measuring 1.9 µg/dl (normal range, 0.5 to 6) and a poststimulation level of 9.5 µg/dl (normal range, 8 to 20). Repeated courses of doxycycline had been given because of repeated episodes of lethargy, depressed appetite, and periodic vomiting and diarrhea despite progressively declining *Ehrlichia* titers, which eventually became negative 1 year before the current evaluation. Over the past 2 years, gastrointestinal signs recurred along with episodes of hypoglycemia The recurrences responded to symptomatic treatment but relapsed with the stress of search training. Most recently, the dog had been hospitalized 1 month earlier for gastroenteritis, which responded to parenteral fluid therapy and antibiotic treatment. Endoscopic biopsies were obtained at that time, and the results from the gastric sample revealed "gastritis—lymphoplasmacytic and neutrophilic, chronic, locally extensive and marked, with gastric mucosal atrophy." The colonic histopathologic evaluation showed "colitis—lymphoplasmacytic, chronic, minimal." This was subsequently treated with oral fenbendazole; no glucocorticoids were given to this dog at any time before this presentation and there was no pertinent travel history.

Physical Examination. The dog was quiet but alert. Temperature, pulse, and respiration rates were normal (101° F, 96 BPM, 12 BPM, respectively). Pulses were palpably weak, oral mucous membranes were pale, and capillary refill time was prolonged, all indicating hypoperfusion. Skin turgor was normal, but the oral mucosa was dry. Rectal examination detected hematochezia.

Diagnostics. CBC, chemistry panel, urinalysis, and fecal examination were performed. The pertinent test results are shown at left.

ASK YOURSELF ...

- What are the problems identified thus far?
- What are the differential diagnoses for the repeated gastrointestinal signs?
- What are your rule-outs for the onset of fatigue associated with field training?
- What are the significant laboratory test abnormalities in this patient, and what are your differential diagnoses for these abnormalities?
- What additional diagnostics would you perform at this time?

ACTH = adrenocorticotrophic hormone

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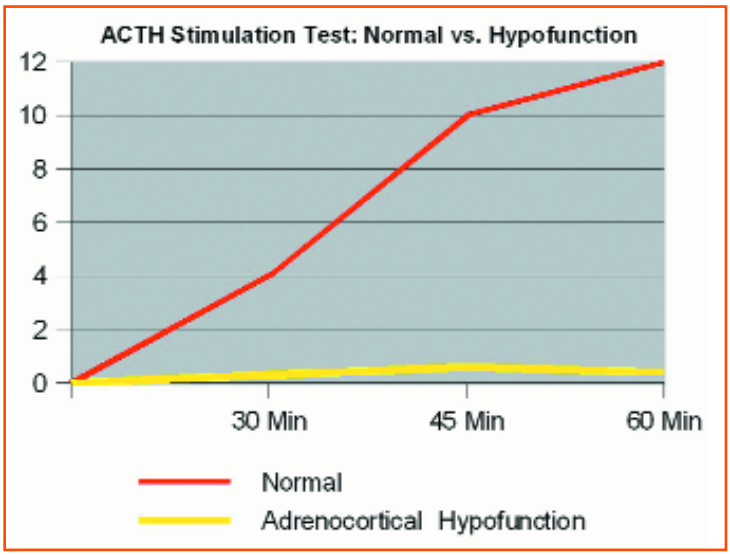
Diagnosis. Atypical hypoadrenocorticism with coexisting IBD.

Adrenocortical function was evaluated with the ACTH-stimulation test (**Chart**). Cosyntropin 0.25 mg was given intramuscularly; blood samples were taken at baseline and 60 minutes later. Resting serum cortisol level was 0.0 µg/dl and remained unchanged in the poststimulation sample. Glucocorticoids were withheld in order to repeat the test the following day using the intravenous route for the cosyntropin (0.25 mg). At this time, resting cortisol was 0.1 µg/dl and the poststimulation cortisol was 0.2 µg/dl, thus confirming the diagnosis of atypical hypoadrenocorticism.



Interpretation. This dog's clinical diagnosis was IBD with coexisting atypical hypoadrenocorticism evidenced by hypocortisolemia accompanied by normal serum sodium and potassium levels. In the absence of prior glucocorticoid use, the hypofunctioning adrenal gland in this dog was attributed to a primary glucocorticoid deficiency due to 1) a primary pathologic condition involving either the zona fasciculata and reticularis of the adrenal cortex or 2) dysfunction involving the synthesis or release of ACTH from the anterior pituitary. The gastrointestinal disease may have reflected primary IBD or may have been related to hypocortisolemia.

Classic canine hypoadrenocorticism is clinically characterized by a history similar to this patient's, along with the laboratory abnormalities of hyponatremia and hyperkalemia as well as hypocortisolemia that is unresponsive to ACTH stimulation. In the absence of the low serum sodium and elevated serum potassium level on biochemical screening tests, many clinicians may not consider hypoadrenocorticism in their differential diagnoses.

This dog had "atypical" hypoadrenocorticism (Addison's disease) as indicated by low serum cortisol levels with coexisting eunatremia and eukalemia.[1-3] The laboratory tests confirmed a hypofunctioning zona fasciculata and reticularis causing hypocortisolemia, which could occur from inadequate ACTH production from a malfunctioning anterior pituitary (secondary hypoadrenocorticism) or autoimmune destruction of the cortisol-producing part of the adrenal cortex.[4] Confirmation of the former would have required measuring the endogenous ACTH concentrations while the latter could have been diagnosed using an indirect immunofluorescent antibody test.[4] Neither test was performed in this patient as treatment for hypoadrenocorticism and IBD resolved the problems.

See Aids & Resources, back page, for references, contacts, and appendices.

DID YOU ANSWER ...

- Prior episodes of vomiting and diarrhea, exercise intolerance, hypoglycemia. Recent onset of lethargy and anorexia accompanied by dehydration, hematochezia, and hypoglycemia
- Inflammatory or other type of infiltrative bowel disease, chronic pancreatitis, intestinal parasites, dietary indiscretion, and metabolic disease
- *Differentials:* Cardiovascular disease (including heartworm disease), pulmonary disease, anemia, neuromuscular disease, metabolic disease, liver disease (in light of low glucose and cholesterol [also compatible with hypoadrenocorticism])
- *Lab Abnormalities:* Leukocytosis possibly due to enteric inflammatory response; lymphocytosis possibly due to cortisol depletion; anemia most likely due to chronic disease; hypoproteinemia due to bowel protein loss in light of the dog's history and absence of other liver abnormalities; hypoglycemia—in this clinical setting may be due to enterotoxemia or hypoadrenocorticism; hypocholesterolemia due to decreased appetite, hypoadrenocorticism, or malnutrition; mild azotemia most likely due to prerenal hypoperfusion; and decreased total CO_2 due to metabolic acidosis
- An ACTH-stimulation test to rule out adrenocortical hypofunction and bile acids to rule out liver disease.

ACTH = adrenocorticotrophic hormone; IBD = inflammatory bowel disease