Bandit: A hypoadrenocorticism (Addison's disease) case study

Patient

Bandit, 2-year-old, neutered male Plott hound mix

Presenting reason

Vomiting, straining to defecate, anorexia, and lethargy

History

Bandit's owners have had him since he was a puppy. He has had a history of a foreign body that did not require surgery and was hit by a car a year ago, but no known dietary indiscretion recently other than a foreign baby bird carcass a week ago.

Bandit was up all night 2 nights ago asking to go outside. Yesterday, he vomited both breakfast and dinner. His owner noted him straining to defecate multiple times with no output. Today he is very lethargic and not interested in food.

Physical examination

Bandit was quiet, alert, and responsive with a body condition score of 5/9. His temperature, pulse, and respiratory rate were within normal limits. Bandit was mildly dehydrated with pink and tacky mucous membranes. His abdomen was soft, nonpainful, and without palpable abnormalities. He had mild scabs on the internal surface of the pinnae, but the rest of the physical exam was within normal limits.

Diagnostic plan

A brief blood screen and radiographs were performed to rule out a foreign body and assess hydration and electrolytes. Radiographs showed no evidence of gastric or small intestinal foreign material or obstruction, and the brief blood screen showed elevated renal values, normal glucose and lactate, and an electrolyte imbalance of low sodium and chloride with normal potassium. Differential diagnoses at this time included toxin, (UTI; less common in males), leptospirosis, and Lyme nephritis due to the geographical location. Hospitalization for intravenous fluid therapy and additional diagnostics were recommended, including a complete blood count (CBC), comprehensive chemistry panel with electrolytes, and complete urinalysis.

Diagnostic review

Bandit's CBC revealed a slightly decreased reticulocyte hemoglobin (RETIC-HGB) without anemia. Decreased RETIC-HGB reflects decreased iron availability for recent red blood cell production. The most common causes are blood loss or inflammatory conditions. In Bandit's case, he also had a significant leukocytosis with neutrophilia. This can be due to inflammation, infection, stress, and other conditions. Bandit also had a mild eosinophilia, which could have been caused by a parasitic or fungal infection, allergic response, or endocrine disease.

A moderate azotemia with elevated phosphorus in Bandit's chemistry panel were noted and concerning for renal damage versus dehydration. Given the history of vomiting, the decreases in sodium and chloride may have been due to gastrointestinal loss. The rest of his chemistry panel was within normal limits.

Hematology

Hematology	5/14/23 4:30 PM		9/13/22 8:41 PM
RBC	7.24	5.65 - 8.87 M/µL	7.59
Hematocrit	43.8	37.3 - 61.7 %	50.6
Hemoglobin	16.8	13.1 - 20.5 g/dL	17.8
MCV	60.5	61.6 - 73.5 fL	66.7
MCH	23.2	21.2 - 25.9 pg	23.5
MCHC	38.4	32.0 - 37.9 g/dL	35.2
RDW	17.0	13.6 - 21.7 %	17.4
% Reticulocyte	0.6	%	0.8
Reticulocytes	44.2	10.0 - 110.0 K/µL	58.4
Reticulocyte Hemoglobin	21.9	22.3 - 29.6 pg	23.8
WBC	26.33	5.05 - 16.76 K/µL	16.79
% Neutrophils	75.4	%	77.8
% Lymphocytes	14.5	%	13.6
% Monocytes	4.0	%	4.3
% Eosinophils	6.0	%	4.3
% Basophils	0.1	%	0.0
Neutrophils	19.85	2.95 - 11.64 K/µL	13.05
Lymphocytes	3.83	1.05 - 5.10 K/µL	2.29
Monocytes	1.05	0.16 - 1.12 K/µL	0.73
Eosinophils	1.57	0.06 - 1.23 K/µL	0.72
Basophils	0.03	0.00 - 0.10 K/µL	0.00
Platelets	259	148 - 484 K/µL	311

Chemistry

Chemistry	5/14/23 4:39 PM		9/13/22 8:49 PM
Glucose	96	74 - 143 mg/dL	142
Creatinine	2.3	0.5 - 1.8 mg/dL	1.0
BUN	39	7 - 27 mg/dL	19
BUN: Creatinine Ratio	17		18
Phosphorus	7.8	2.5 - 6.8 mg/dL	2.4
Calcium	11.1	7.9 - 12.0 mg/dL	10.5
Sodium	137	144 - 160 mmol/L	154
Potassium	5.6	3.5 - 5.8 mmol/L	3.8
Na: K Ratio	24		41
Chloride	105	109 - 122 mmol/L	115
Total Protein	6.9	5.2 - 8.2 g/dL	6.1
Albumin	3.2	2.3 - 4.0 g/dL	3.0
Globulin	3.8	2.5 - 4.5 g/dL	3.1
Albumin: Globulin Ratio	0.8		1.0
ALT	99	10 - 125 U/L	- --.--
ALP	68	23 - 212 U/L	49
GGT	0	0 - 11 U/L	7
Bilirubin - Total	0.2	0.0 - 0.9 mg/dL	0.5
Cholesterol	150	110 - 320 mg/dL	223
Amylase	1,254	500 - 1,500 U/L	616
Lipase	521	200 - 1,800 U/L	832
Osmolality	283	mmol/kg	308

Addison's Disease Risk

Insight ***created on May 14, 2023.***

Has this dog been on any recent systemic or topical medications in the last 1-3 months which might affect cortisol production?

- (●) No, no recent medications that impact cortisol production
- ( ) Yes, treatment for Cushing's disease
- ( ) Yes, recent glucocorticoid medications

Does this dog have one or more clinical signs consistent with hypoadrenocorticism (Addison's Disease)?

- (●) Yes, Addison's clinical signs present
- ( ) No, Addison's clinical signs not present

There are patterns present in this patient's recent bloodwork that are concerning for possible Addison's disease.

IDEXX DecisionIQ™ Addison's disease risk

Patterns present in Bandit's bloodwork results triggered an IDEXX DecisionIQ Addison's disease risk indicator.

The IDEXX DecisionIQ Addison's disease risk indicator is a machine-learning artificial intelligence model developed to support clinicians by identifying dogs that are at an increased likelihood of Addison's disease and that would benefit from targeted screening. Based on the abnormal electrolytes, azotemia, and clinical signs, IDEXX DecisionIQ recommended a resting cortisol with ACTH stimulation test if indicated. A Fecal Dx® Antigen Panel was also recommended if appropriate to rule out whipworm-associated pseudo-Addison's disease as a possible cause of the electrolyte changes.

Next steps and results

Bandit's emergency veterinary team had Addison's disease on their differential list but was grateful to see the alert supported their suspicion. A baseline SNAP® Cortisol Test was added to the bloodwork in the hospital as a next screening step for Bandit. The results showed that Bandit had low levels of circulating cortisol. Based on these results, a follow-up ACTH stimulation was performed next. Bandit's adrenal glands showed no response to the ACTH with both the pre-ACTH and post-ACTH results below the level of detection.

Resting cortisol

Endocrinology	5/14/23 5:12 PM	
Cortisol - Baseline	<0.5	µg/dL

ACTH stimulation test

Endocrinology	5/15/23 2:15 PM	
Cortisol - Pre ACTH	<0.2	µg/dL
Cortisol - Post ACTH	a. 0.2	µg/dL

a. ACTH Reference Range:

Canine:	Feline	
2-6	0.5-5	Pre-ACTH (resting) cortisol
6-18	5-15	Post-ACTH cortisol
18-22	15-19	Equivocal post-ACTH cortisol
>22	>19	Post-ACTH cortisol consistent with hyperadrenocorticism
<2	<0.5	Post-ACTH cortisol consistent with hypoadrenocorticism

Diagnosis

Bandit was diagnosed with Addison's disease based on the results of the ACTH stimulation test.

Follow-up

Bandit was started on 1.5 tablets of 10 mg prednisone orally in a tapering dose for glucocorticoid replacement and was given an initial injection of desoxycorticosterone pivalate (DOCP) for mineralocorticoid replacement therapy.

Bandit has been doing well at home and is maintained on lowest effective dosing of prednisone. His recheck electrolytes have been stable and will continue to be monitored as his referring DVM is monitoring for optimal DOCP injection intervals.

Bandit's owner reports he has "come back to life" after starting treatment for Addison's disease and feels he was sick some time prior to his diagnosis. His formerly coarse coat has softened again, and Bandit's owner is very pleased with the prompt diagnosis and excellent care.

Recheck chemistry

Chemistry	6/9/23 2:55 PM		5/24/23 9:45 AM
Sodium	151	144 - 160 mmol/L	148
Potassium	4.2	3.5 - 5.8 mmol/L	4.7
Na: K Ratio	36		31
Chloride	112	109 - 122 mmol/L	111

Discussion

Hypoadrenocorticism, also known as adrenal insufficiency or Addison's disease, is a condition that results from a bilateral dysfunction of the adrenal cortex, or outer layer of the adrenal glands. The adrenal cortex produces important hormones including aldosterone (mineralocorticoid), cortisol (glucocorticoid), and other adrenal androgens.

The typical representation of Addison's disease is a deficiency of both glucocorticoids and mineralocorticoids. These hormones are crucial for the regulation of heart rate, blood glucose, blood pressure, electrolyte balance, digestion, and maintaining homeostasis in response to physical and mental stress. CBC abnormalities in patients with Addison's disease can show mild nonregenerative anemia, eosinophilia, and an absence of a stress leukogram.

Chemistry panel abnormalities can include azotemia, hypocholesterolemia, hypoglycemia, hypoalbuminemia, hypercalcemia, hyperkalemia, hypochloremia, hyponatremia, low Na:K ratio, and elevated liver enzymes. Abnormal electrolytes due to mineralocorticoid deficiency are among the more recognizable signs of Addison's disease. However, they are seen inconsistently in Addison's disease and may only be present with more advanced disease or when a dog is in crisis.

Atypical primary Addison's disease cases with isolated glucocorticoid deficiency are rare. Most cases initially presenting as glucocorticoid-deficient will only go on to develop electrolyte abnormalities requiring mineralocorticoid supplementation within 3 months of initial diagnosis.

Most cases have a history of vague, intermittent clinical signs that can include ain't doing right (ADR), recurrent digestive upset, lethargy, weight loss, increased urination, and decreased appetite. These signs may worsen in association with stressful events. Some may present in crisis with collapse, hypovolemia, and bradycardia. Both the nonspecific or intermittent GI signs and more subtle patterns seen within a CBC and chemistry panel in earlier Addison's disease can be mistaken for more common diseases.

About 30% of the time, Addison's disease is diagnosed after the dog has experienced an Addisonian crisis. These crises can be severe, requiring hospitalization. Left untreated or without rapid intervention, Addison's disease can be fatal.*

References:

AAHA Endocrinology Guidelines, Bugbee A, Rucinsky R, et al. 2023 AAHA Selected Endocrinopathies of Dogs and Cats Guidelines. J Am Anim Hosp Assoc. 2023; 59 doi 10.5326/JAAHA-MS-7368.

*Lathan P, Thompson AL. Management of hypoadrenocorticism (Addison's disease) in dogs. Vet Med (Auckl). 2018 Feb 9;9:1-10. doi: 10.2147/VMRR.S125617. PMID: 30050862; PMCID: PMC6055912.