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Atypical hypoadrenocorticism in a young adult dog

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Abstract: This case report presents a young adult dog with atypical hypoadrenocorticism. A 2-year-old dog presented with nausea, vomiting, lethargy and poor appetite for a day. At the initial physical examination, the body temperature was normal, a respiratory rate of 45 and a heart rate of 80 bpm without audible cardiac murmurs. Ultrasound examination showed a slightly enlarged gastric wall with decreased motility and no other abnormalities. Hematological and biochemical blood profiles were unremarkable. Based on these findings a treatment for acute gastritis was initiated. Two days later, the dog presented with vomiting, decreased appetite and profound weakness. At the physical examination, the heart rate was 51 bpm, the respiratory rate was 48 and no abdominal discomfort. Routine serum biochemical profile was unremarkable. An ACTH stimulation test was performed and fluid therapy and dexamethasone were instituted with rapid clinical improvement. Pre and post ACTH stimulation test values were 0,05 µg/dL and 5,15 µg/dL, respectively. Because the first ACTH stimulation test was inconclusive, a second ACTH stimulation test was repeated one month later, when cortisol values confirmed the presence of atypical hypoadrenocorticism syndrome.

Introduction

Naturally occurring hypoadrenocorticism (Addison's disease) is an uncommon illness, with estimates of its incidence ranging from 0.36% to 0.5%. The clinical syndrome occurs when at least 85% to 90% of the adrenocortical tissue is destroyed, resulting in deficiencies of mineralocorticoids and glucocorticoids. Atypical hypoadrenocorticism represents a minority (10%) of primary hypoadrenocorticism patients that have normal serum electrolytes at initial diagnosis. This may occur secondary to gradual loss of adrenocortical tissue in which loss of glucocorticoid-secreting portions precedes loss of the mineralocorticoid-secreting layer of the adrenal cortex. These patients may go on to develop electrolyte abnormalities in days to months following initial diagnosis. Naturally occurring secondary hypoadrenocorticism results from failure of the pituitary gland to secrete ACTH. Lack of ACTH leads to severe atrophy of the adrenal zona fasciculata and the zona reticularis, with an intact zona glomerulosa. Serum electrolytes remain normal because aldosterone secretion is preserved. Hypoadrenocorticism can affect dogs of any age, but it tends to occur in young to middle-aged dogs. The age range of reported cases is 4 wk to 16 y. The average age at diagnosis is 4 to 5 y. Most studies suggest that female dogs account for the majority of patients with Addison's disease, at 69%. Addison's disease occurs in many breeds, but certain breeds appear to have an increased risk for developing this syndrome. These breeds include great danes, poodles (all types), west highland white terriers, Portuguese water dogs, bearded collies, rottweilers, soft-coated wheaten terriers, springer spaniels, bas-sett hounds, and Nova Scotia duck tolling retrievers. There are no pathognomonic clinical signs for hypoadrenocorticism. Rather, Addison's disease causes vague and nonspecific clinical signs that can be attributed to multiple body systems and diseases, including gastrointestinal disease, renal failure, or neurological disease. Given this, definitive diagnosis and prompt treatment rests a great deal on the clinician's index of suspicion that prompts an ACTH stimulation test. This is particularly true in secondary or atypical hypoadrenocorticism, as there may be few laboratory abnormalities to help guide further diagnostics and therapeutics.

Material and method

A 2-year-old castrated male Poodle presented with an history of 12 hours of nausea, vomiting, lethargy and poor appetite for a day. At the initial physical examination, the body temperature was normal, a respiratory rate of 45 and a heart rate of 90 bpm without audible cardiac murmurs. Ultrasound examination showed a slightly enlarged gastric wall with decreased motility and no other abnormalities. The blood analysis were normal. Two days later, the dog presented with abnormal physical examination findings, generalized muscle weakness, slight decreased mentation, poor peripheral pulses, capillary refill time (CRT) > 2 s, bradycardia (60 bpm), a normal temperature (37.7°C) and slight abdominal pain. Blood work was initiated prior any treatment with unremarkable results. Despite the age of the dog, hypoadrenocorticism was suspected and intravenous crystalloid fluid therapy (Lactate Ringer, B. Braun) and glucose 5% was initiated. Dexamethasone (0.25 mg/kg IV) was administered, and an ACTH stimulation test (250 ug IV) was performed.

Results and discussions

Cell blood count was unremarkable (table 1), all the parameters were in referance range except MCH and MCHC which were slight increased. The biochemical profile showed a slight decreased of total proteins and globulin concentrations (table 2).

Parameter	Value	Parameter	Value
Erytrocytes (M/µL)	7.05	Platelets (K/µL)	192
Leucocytes (K/µL)	8.31	Reticulocytes (K/µL)	56.7
Hemoglobin(g/dL)	17.8	Neutrophils (K/µL)	5.04
Hematocrit(%)	47.1	Lymphocytes (K/µL)	2.37
MCV(fL)	66.8	Monocites (K/µL)	0.66
MCH (pg)	25.3	Eoshinophils (K/µL)	0.14
MCHC ((g/dL)	37.8	Basophils (K/µL)	0.05
RDW (%)	11.3		

Table 1. Hematological blood parameters

Parameter	Value	Parameter	Value
Creatinine (mg/dl)	0.68	Total bilirubin (mg/dl)	0.06
Ureea (mg/dl)	40	Lipase (U/I)	31.91
TGP (U/I)	33	Amylase (U/I)	890
TGO (U/I)	25	Na+ (mmol/L	153.85
GGT (U/I)	2	K+ (mmol/L)	4.08
ALP (U/I)	14.02	total proteins (g/dl)	4.9
Glucose (mg/dl)	95.48	Albumin (g/dl)	3.73
Triglycerids (mg/dl)	98	Globulins (g/dl)	1.2

Table 2. Biochemical serum profile

- Pre and post ACTH stimulation test values were 0,05 µg/dL and 5,15 µg/dL, respectively. The test was inconclusive, the values obtained didn't exclude Adisson disease. More than that, the dog responded to fluid therapy and corticosteroids with a rapid clinical improvement.
- Because the first ACTH stimulation test was inconclusive, a second ACTH stimulation test was repeated one month later. Pre and post second ACTH stimulation test were 0.06 µg/dL and 1.8 µg/dL , respectively.
- Also routine hematological and biochemical profiles were performed prior testing. All the parameters were in the reference range.

Conclusions

- Hypoadrenocorticism syndrome could be included on the differential list in young adult dogs with gastroenteric symptoms.
- Gastrointestinal signs and bradycardia are symptoms common in hypoadrenocorticism.
- In atypical hypoadrenocorticism the routine hematological and biochemical profiles could be inconclusive.
- ACTH stimulation test represents the gold standard method in diagnosis of hypoadrenocorticism.