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Hypoadrenocorticism in Dogs

Hypoadrenocorticism (Addison's disease) is an uncommon disease in dogs. Young to middle-aged females and Great Danes, standard poodles, and West Highland white terriers are predisposed, but any dog can be affected. Because of the potential for acute death in dogs with severe electrolyte abnormalities and the favorable prognosis with appropriate treatment, prompt diagnosis is crucial.

Hypoadrenocorticism is caused by a lack of corticosteroid and mineralocorticoid secretion from the adrenal glands. Common clinical signs include inappetence, vomiting, weight loss, and diarrhea. Hematemesis and hematochezia occur less frequently. Dogs often have waxing and waning clinical signs that resolve with fluid or corticosteroid administration. If diagnosis is delayed, patients may present in hypovolemic shock.

Diagnosis

The classic laboratory findings in hypoadrenocorticism are hyponatremia and hyperkalemia, although these findings may be absent initially. Some patients with hypoadrenocorticism ("atypical" hypoadrenocorticism) do not exhibit characteristic electrolyte alterations. Other conditions, such as renal disease and gastrointestinal parasitism, may also cause hyperkalemia and hyponatremia. Other laboratory abnormalities in patients with typical hypoadrenocorticism include hypoglycemia, hypochloremia, hypocholesterolemia, and azotemia. Most addisonian patients present with a urine specific gravity less than 1.030; in the face of azotemia, this may be incorrectly attributed to primary renal failure. Eosinophilia, lymphocytosis, or lack of a stress leukogram in an ill patient suggests hypoadrenocorticism.

Definitive diagnosis requires an ACTH stimulation test. A post-ACTH plasma cortisol concentration less than 2 µg/dl is consistent with hypoadrenocorticism. A single dose of dexamethasone given before the ACTH stimulation test does not interfere with interpretation of results.

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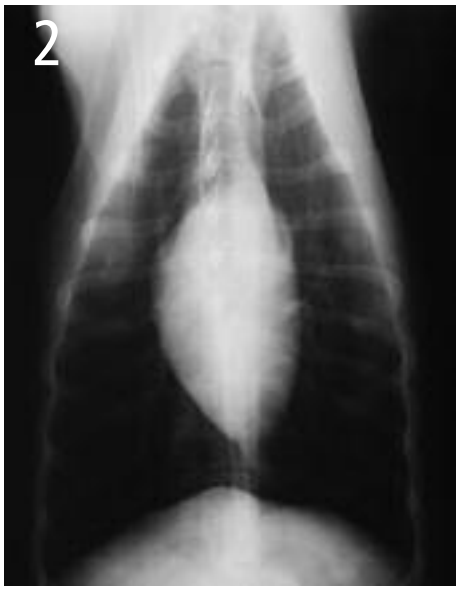
A New Column

Patients sometimes require treatment and monitoring that is difficult to provide in the generalist's clinic. However, referral is often simply not an option. This column, a replacement of our former "How to Refer" series, provides information about when to consider referring, how to do so most effectively, and how to manage the patient when referral is impractical or impossible.

This dog presented for acute collapse while hunting. Laboratory findings included hyponatremia, hyperkalemia, azotemia, and severe hypoglycemia. After initial therapy with intravenous fluids and dextrose, hypoadrenocorticism was confirmed with an ACTH stimulation test. With desoxycorticosterone pivalate and prednisone supplementation, the dog returned to his normal routine, including additional hunting trips.



Thoracic radiograph from a dog with hypoadrenocorticism revealing microcardia. Microcardia is a nonspecific finding indicative of hypovolemia.

ACTH = adrenocorticotropic hormone

Levels of Care

Hemodynamically stable patients with mild electrolyte abnormalities (potassium < 5.5 mEq/L, sodium > 135 mEq/L) and clinical signs may be managed as outpatients. Prednisone is administered at supraphysiologic doses initially (0.5 to 1.0 mg/kg per day) and then adjusted to the lowest effective dose; the dose should be doubled the day before anticipated stressful situations. For mineralocorticoid supplementation, desoxycorticosterone pivalate (DOCP; 2.2 mg/kg) is administered as an intramuscular or subcutaneous (if well-hydrated) injection every 25 days. Alternatively, oral fludrocortisone acetate may be given at a starting dosage of 0.02 mg/kg per day. Fludrocortisone also has corticosteroid activity, so additional prednisone may not be necessary.

When to Consider Referring

Patients with severe clinical signs and laboratory abnormalities must be stabilized with intravenous fluid therapy. Referral of patients with severe electrolyte abnormalities, acid–base disturbances, gastrointestinal blood loss, or other metabolic abnormalities is recommended for intensive monitoring and therapy. These patients are best handled by facilities with 24-hour care and the ability to frequently monitor electrolytes, blood pressure, and ECGs. Access to blood products is ideal.

When Referral is Not an Option

The primary goals in management of an addisonian crisis are to correct hypovolemia, electrolyte abnormalities, and hypoglycemia (if present). Blood pressure, ECGs, and electrolytes should be monitored frequently. Hyperkalemia usually improves with fluid administration alone. The intravenous fluid of choice is 0.9% sodium chloride, administered at shock doses (90 ml/kg, given as 20- to 30-ml/kg boluses). For hypoglycemia, dextrose can be administered as a diluted bolus or a 5% solution in 0.9% sodium chloride.

If hyperkalemia is severe (potassium > 6.5 mEq/L) or causing ECG changes (absent P waves, increased P-R interval, or bradycardia), additional therapy may be indicated. A 10% solution of calcium gluconate (2 to 10 ml/dog) may be administered intravenously over 10 to 15 minutes while monitoring for ECG changes associated with hypercalcemia. Although the effect is almost immediate, it lasts for only about 10 to 30 minutes; the treatment is cardioprotective and does not lower the potassium concentration. Simultaneous intravenous administration of dextrose (2 g/unit of insulin) and regular insulin (0.5 U/kg) will decrease potassium levels within 15 to 30 minutes. A 5% dextrose solution in 0.9% sodium chloride should be administered after insulin treatment to alleviate hypoglycemia.

Following stabilization, an ACTH stimulation test should be performed. Dexamethasone (0.2 mg/kg intravenously) may be administered before or after ACTH stimulation because, unlike prednisone, it will not interfere with interpretation. Fluid therapy should continue until the patient can eat and drink. Additional supportive therapy, including gastroprotectants and blood transfusion, may be necessary. Dexamethasone should be given daily (0.05 mg/kg Q 12 H) until oral prednisone is tolerated. Mineralocorticoid supplementation should begin once the diagnosis is confirmed.

The Referral Process

Initial stabilization must be attempted before referral because the stress of transportation may not be tolerated. Intravenous fluids should be given as described above, and hyperkalemia and hypoglycemia should be controlled.

Recent laboratory values, radiographs, and medical records (including a list of medications) should be sent with the patient. The client should be informed that although most addisonian patients recover fully, lifelong medication is required. The cost for an addisonian patient is approximately $2 to $3/kg per month.

Findings in Dogs with Hypoadrenocorticism

Historical
Decreased appetite
Lethargy
Weight loss/failure to gain weight
Depression/weakness
Vomiting
Diarrhea
Hypovolemia/shock
Polyuria/polydipsia
Shaking/shivering
Physical examination
Depression/weakness
Hypovolemia/shock/collapse
Bradycardia
Melena
Abdominal pain
Clinicopathologic
Hyperkalemia
Hyponatremia
Hypochloremia
Azotemia
Hypoglycemia
Hypercalcemia
Hypocholesterolemia
Hypoalbuminemia
Isosthenuria
Lymphocytosis
Eosinophilia
Lack of stress-related neutropenia
Anemia

Following discharge, follow-up laboratory monitoring and dosage adjustments are required, and the referral clinic should be contacted about questions regarding dose adjustment or treatment response. Although addisonian patients have an excellent long-term prognosis, concurrent illness may affect long-term management. Thus, reconsultation with a specialist should be considered when new problems are identified.

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

ACTH = adrenocorticotropic hormone; ECG = electrocardiogram