

HYPOADRENOCORTICISM OR ADDISON'S DISEASE

(INADEQUATE PRODUCTION OF HORMONES BY THE ADRENAL GLANDS)

BASICS

OVERVIEW

- A hormonal disorder resulting from decreased production of hormones (glucocorticoids and/or mineralocorticoids) by the adrenal glands
- “Glucocorticoids” are a class of hormones produced by the adrenal glands; they typically are called “steroids;” glucocorticoids are involved in metabolism and the stress response and they have anti-inflammatory properties
- “Mineralocorticoids” are another class of hormones produced by the adrenal glands; they are involved in regulation of salt (sodium and potassium) in the body; “aldosterone” is a mineralocorticoid that regulates sodium and potassium in the body
- Addison’s disease refers to decreased production of both glucocorticoids and mineralocorticoids
- Glucocorticoid (cortisol) deficiency contributes to lack of appetite (known as “anorexia”); vomiting; black, tarry stools (due to the presence of digested blood; condition known as “melena”); sluggishness (lethargy); and weight loss
- Inadequate glucocorticoid levels increase likelihood of the patient developing low blood glucose or sugar (known as “hypoglycemia”)
- Mineralocorticoid (aldosterone) deficiency results in an inability to retain sodium in the body and to excrete potassium from the body; decreased sodium levels lead to diminished circulating blood volume that in turn contributes to low blood pressure (known as “hypotension”), dehydration, weakness, and depression; increased levels of potassium in the blood (known as “hyperkalemia”) may result in heart-muscle toxicity

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Great Danes, rottweilers, Portuguese water dogs, standard poodles, West Highland white terriers, and soft-coated wheaten terriers have increased risk as compared to other dog breeds
- No breed predilection in cats

Mean Age and Range

- Dogs—range, less than 1 year to greater than 12 years of age; median, 4 years of age
- Cats—range, 1 to 9 years of age; most are middle-aged

Predominant Sex

- Female dogs are more likely to have hypoadrenocorticism than male dogs
- No predominant sex in cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vary from mild and few in some patients with long-term (chronic) low levels of steroids produced by the adrenal glands (hypoadrenocorticism) to severe and life-threatening disease in a sudden (acute) Addisonian crisis (condition in which patient is in shock and collapse, usually with low body temperature [known as “hypothermia”], weak pulse, and an unexpectedly slow heart rate)
- Dogs—sluggishness (lethargy); lack of appetite (anorexia); vomiting; weight loss; signs vary in intensity—they may increase and decrease over time (known as a “waxing and waning” course); diarrhea; shaking; increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”)
- Dogs—depression; weakness; dehydration; collapse; low body temperature (known as “hypothermia”); black, tarry stools (melena); weak pulse; slow heart rate (known as “bradycardia”); painful abdomen; hair loss
- Cats—sluggishness (lethargy); lack of appetite (anorexia); vomiting; increased urination (polyuria) and increased thirst (polydipsia); weight loss
- Cats—dehydration; weakness; weak pulse; slow heart rate (bradycardia)

CAUSES

- Primary hypoadrenocorticism—unknown cause (so called “idiopathic disease”); immune-mediated disease; side effect of medication (mitotane) used to treat excessive production of steroids by the adrenal glands (condition known as “hyperadrenocorticism” or “Cushing’s disease”); cancer
- Secondary hypoadrenocorticism—side effect of medical treatment with steroids, when long-term steroid administration is discontinued; abnormalities in the pituitary gland; the “pituitary gland” is the master gland of the body—it is located at the base of the brain; it controls many other glands in the body

RISK FACTORS

- Treatment for excessive production of steroids by the adrenal glands (hyperadrenocorticism or Cushing’s disease)
- Long-term use of steroids in medical treatment

TREATMENT

HEALTH CARE

- A sudden (acute) Addisonian crisis (condition in which patient is in shock and collapse, usually with low body temperature [hypothermia], weak pulse, and an unexpectedly slow heart rate) is a medical emergency requiring intensive therapy
- Treat sudden (acute) Addisonian crisis with rapid correction of low blood volume (known as “hypovolemia”) using isotonic fluids (preferably 0.9% NaCl)
- Treatment of long-term (chronic) hypoadrenocorticism depends on severity of clinical signs; usually initial stabilization and therapy are conducted on an inpatient basis

ACTIVITY

- No alteration necessary

DIET

- No alteration necessary

MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- In an Addisonian crisis (condition in which patient is in shock and collapse, usually with low body temperature [hypothermia], weak pulse, and an unexpectedly slow heart rate), administration of a rapidly acting glucocorticoid or steroid (such as dexamethasone sodium phosphate or prednisolone sodium succinate) by injection is indicated
- Fluid therapy with 0.9% NaCl as needed, based on the patient’s hydration, volume status and blood pressure
- Long-term (chronic) primary hypoadrenocorticism—treat with adrenal hormone replacement medications (glucocorticoid replacement [prednisone] and mineralocorticoid replacement [desoxycorticosterone pivalate or “DOCP” or fludrocortisone acetate])
- DOCP usually is required at monthly intervals, a few patients need injections every 3 weeks, and rare patients need injections every 2 weeks
- Patients with confirmed secondary hypoadrenocorticism require only glucocorticoid or steroid supplementation (prednisone)

FOLLOW-UP CARE

PATIENT MONITORING

- After the first 2 injections of DOCP, ideally do blood work and measure serum electrolyte (especially sodium and potassium) levels at 2, 3, and 4 weeks to determine duration of effect; thereafter, check electrolyte levels at the time of injection for the next 3 to 6 months (and adjust the dosage of DOCP, if necessary) and then every 6 months
- Adjust the daily dose of fludrocortisone, based on serial blood work (serum electrolyte determinations); following initiation of therapy, check serum electrolyte levels weekly until they stabilize in the normal range; thereafter, check serum electrolyte concentrations and blood urea nitrogen or creatinine monthly for the first 3 to 6 months and then every 3 to 12 months

PREVENTIONS AND AVOIDANCE

- Continue adrenal hormone replacement therapy for the lifetime of the patient
- Increase the dosage of replacement glucocorticoids or steroids during periods of stress (such as travel, hospitalization, and surgery), as directed by your pet’s veterinarian

POSSIBLE COMPLICATIONS

- Increased urination (polyuria) and increased thirst (polydipsia) may occur from prednisone administration, necessitating decreasing or discontinuing the drug
- Increased urination (polyuria) and increased thirst (polydipsia) may occur from fludrocortisone administration, necessitating a change to DOCP therapy

EXPECTED COURSE AND PROGNOSIS

- Most patients carry a good to excellent prognosis following proper stabilization and treatment
- Patients with underlying tumors or cancer have less favorable prognoses

KEY POINTS

- Lifelong glucocorticoid and/or mineralocorticoid replacement therapy is required
- Increased dosages of replacement glucocorticoids or steroids are required during periods of stress (such as travel, hospitalization, and surgery), as directed by your pet’s veterinarian

