

Canine Hypoadrenocorticism
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Canine Hypoadrenocorticism

1. Introduction

A. Etiology

1. Primary adrenocortical failure (Addison's disease)

A. Auto-immune disorder (Most common type)

B. Destructive lesions

1. Histoplasmosis

2. Blastomycosis

3. Metastatic neoplasia

C. Infarction of adrenals

D. Amyloidosis

E. Secondary to o,p'-DDD

2. Secondary (lack of ACTH secretion)

A. Destructive lesions of pituitary/hypothalamus.

B. Chronic, excessive steroid use.

C. Hypopituitarism

D. Idiopathic

2. Signalment

A. Young to middle age.

B. Females > Males (80% Female).

C. May be a breed predilection in Standard Poodles.

3. Pathophysiology

A. Requires 90% loss of adrenal cortex.

B. Destruction is usually gradual and symptoms first appear during times of stress (trauma, surgery, infection). Ultimately, hormone secretion is inadequate even under normal conditions.

C. Lack of glucocorticoids

1. Gastrointestinal effects

A. Anorexia

B. Vomiting

C. Abdominal Pain

D. Weight loss

2. Mental changes

A. Lethargy

3. Metabolic effects

A. Decreased gluconeogenesis.

B. Decreased fat metabolism and utilization.

C. Hepatic glycogen depletion leading to fasting hypoglycemia.

D. Lack of aldosterone

1. Inability to conserve sodium

A. Hypovolemia

B. Weight loss

C. Decreased blood pressure

D. Decreased cardiac output

E. Decreased renal blood flow; pre- renal azotemia

F. Weakness

2. Inability to excrete potassium

A. Due to reduced GFR

B. Decreased excitability of heart

C. Slows conduction (EKG abnormalities)

E. o,p'-DDD therapy can produce similar signs

1. Sodium and potassium should be monitored during therapy.

F. Secondary adrenal insufficiency

1. Aldosterone normal so no electrolyte abnormalities.

2. Signs due to glucocorticoid insufficiency.

4. Clinical Signs

A. Waxing - waning course

B. Lethargy/depression

C. Weakness

D. Anorexia/weight loss

E. Vomiting/diarrhea/melena

F. Abdominal pain

G. PU / PD

5. Physical Examination

A. Weakness/unable to stand.

B. Weak, thready pulse.

C. Dehydration

D. Tachycardia (bradycardia with Hyperkalemia).

E. Abdominal pain

6. Laboratory Abnormalities

A. Hemogram

1. Normocytic, normochromic anemia (may be masked by dehydration). Marked GI blood loss can also occur.
2. Eosinophilia and lymphocytosis. Normal lymphocyte and eosinophil counts in a severely ill animal should make you suspicious of hypoadrenocorticism.

B. Biochemistry profile

1. Uremia

- A. Usually pre-renal
- B. Specific gravity 1.010-1.025
- C. Responds to fluid therapy.
- D. Frequently Addison's misdiagnosed initially as renal failure.

2. Hypoglycemia (rare)

3. Hypercalcemia (25% of cases): Decreased renal calcium excretion.

4. Metabolic acidosis

5. Electrolyte abnormalities

- A. Classically hyponatremia, hypochloremia, hyperkalemia.
- B. May be normal if some aldosterone production is present. The disease can be slowly progressive and is not an "all-or-none" phenomenon.
- C. May be normal if treated elsewhere with fluids.
- D. Will be normal if atypical hypoadrenocorticism or ACTH deficiency (very rare).
- E. May be normal or have hypokalemia with severe intestinal loss (vomiting, diarrhea, GI hemorrhage).
- F. A Na/K ratio < 25:1 suggestive of Addison's.

G. A decreased Na/K ratio may also be seen with:

1. Renal failure
2. Severe acidosis
3. Primary gastrointestinal disease (esp whipworms).
4. Severe liver disease.
5. Pleural and peritoneal effusions.

7. Radiographic Abnormalities

- A. Microcardia
- B. Decreased size of aorta and vena cava.
- C. Megaesophagus (rare)

8. EKG Abnormalities

A. Useful in assessing hyperkalemia and following response to treatment. Not helpful in predicting initial K level. Most helpful if you can measure the potassium at the beginning and use the EKG to monitor response to therapy.

B. Potassium > 5.5

1. Spiked T wave
2. Shortened Q-T interval

C. Potassium > 6.5

1. Prolonged QRS complex

D. Potassium > 7.0

1. Prolonged P wave, decreased amplitude
2. Prolonged P-R interval
3. Prolonged QRS complex, decreased amplitude

E. Potassium > 8.5

1. Lack of P waves

2. Bradycardia
3. Ventricular fibrillation

9. Diagnosis

A. ACTH stimulation test

1. To test adrenal reserve
2. Same as protocol described under hyperadrenocorticism.
3. Pre and post-cortisol below normal basal level.
4. Test can be performed while instituting emergency therapy (see below).

B. Endogenous ACTH concentration

1. To distinguish primary versus secondary hypoadrenocorticism. Mainly of academic interest as the treatment is the same.
2. Elevated with primary disease or post-Lysodren therapy.
3. Decreased with secondary disease or following overdosage with steroids.

10. Treatment

A. Correct hypovolemia

1. 40-80 ml/kg normal saline (0.9%) IV over 1st hour.
2. Decrease to maintenance needs.
3. If concerned about hypoglycemia make solution 5% dextrose in saline.
4. The most important aspect of therapy is fluid replacement.

B. Correct electrolyte abnormalities

1. Fluid therapy is the best therapy.
 - A. Saline restores sodium and chloride concentration.
 - B. Promotes potassium excretion.

C. Corrects hypovolemia

D. Corrects pre-renal azotemia

E. Promotes correction of acidosis by increasing tissue perfusion.

C. Glucocorticoid replacement (also see below)

1. 30 mg/kg IV bolus Solu-Delta-Cortef.

2. 1 mg/kg dexamethasone sodium phosphate IV (place in IV bottle).

D. Acidosis

1. Use arterial blood gas or venous tCO₂ to determine base deficit.

2. Re-check following first hour of therapy. If tCO₂ still <12, administer bicarbonate by adding it to the maintenance fluids. Calculate bicarb need: mEq bicarb = Body weight (kg) X 0.5 X Base deficit. Only add 25% of this total to the fluids and recheck in 4 hours. It is rarely necessary to use bicarbonate as most of these dogs will normalize or increase their tCO₂ following fluid therapy.

E. Obtaining a diagnosis

1. The ACTH stimulation test can be performed by taking the pre-ACTH cortisol sample when blood is obtained for other lab work (BUN, electrolytes, glucose, CO₂, etc).

2. The ACTH is injected and the post sample is obtained 1-2 hours later depending on your protocol.

3. Treatment with Solu-Delta-Cortef, can be started following completion of the test. Dexamethasone can be used simultaneously as it will not interfere with the cortisol assay.

11. Maintenance Therapy

A. Mineralocorticoid therapy

1. Start when animal is eating and drinking again.

2. Fludrocortisone acetate (Florinef).

3. 0.10 mg/10 lb starting dose.

4. Maintain K concentration between 4.0-5.5 mEq/L.

5. Monitor electrolytes and BUN every 1-2 weeks initially, then every 3-4 months when stable.

6. If K is still high, increase Florinef by 0.10 mg/day and re-check in one week.

7. Can also use desoxycorticosterone pivalate (DOCP) 2.2 mg/kg IM or SQ every 21-25 days.

B. Glucocorticoid therapy

1. May not be needed in all dogs.

2. May need replacement dose to prevent signs of glucocorticoid deficiency (0.2-0.4 mg/kg/day of prednisone).

1. Should give owner 5 mg prednisone pills to give to the dog during illnesses or stress (hospitalization, boarding, etc.)