Article #4

Feline Hypoadrenocorticism

Bronya Redden, DVM*
East Lincoln Animal Hospital
Denver, North Carolina

ABSTRACT:

Feline hypoadrenocorticism is a rare disease but should be considered in patients with vague clinical signs and electrolyte imbalances. This article reviews the cause, clinical signs, typical blood work abnormalities, diagnosis, and treatment of feline hypoadrenocorticism. A case report of a 2½-year-old castrated domestic shorthaired cat diagnosed with hypoadrenocorticism is also presented. The initial hypoadrenal crisis was managed in the hospital with intravenous fluids, dexamethasone, and repository intramuscular desoxycorticosterone pivalate. Chronic management consisted of mineralocorticoid support with repository intramuscular desoxycorticosterone pivalate every 25 to 30 days and periodic glucocorticoid support with oral prednisolone. Serum electrolytes were monitored periodically. Three years after diagnosis, the cat continues to be well managed and has an excellent long-term prognosis with continued treatment.

Relatively few cases of feline hypoadrenocorticism have been reported in the literature.[1–7] Since the first case was reported in 1983, only 22 have been published.[4,8] Currently, the pathogenesis of feline primary hypoadrenocorticism remains unknown.[1,6,8] Primary hypoadrenocorticism results from destruction of the adrenal cortex and consequent inadequate production of glucocorticoids and mineralocorticoids.[1,3,8] Secondary hypoadrenocorticism is a result of inadequate production of adrenocorticotropic hormone (ACTH) by the pituitary and has not been reported in cats.[3,4] However, suppression of ACTH production can be caused by glucocorticoid and progestogen administration[3]; high doses of methylprednisolone acetate and recommended doses of megestrol acetate have been shown to suppress the plasma cortisol response to ACTH and cause adrenal cortex atrophy in cats.[3]

In dogs, primary hypoadrenocorticism is considered idiopathic but is most likely the result of autoimmune destruction of the adrenal cortex.[1,2,9] In one case report, necropsy was performed on a cat diagnosed with hypoadrenocorticism; the results showed lymphocytic infiltration of the adrenal cortex, suggesting immune-mediated destruction of the adrenal cortex.[2] Currently, most cases of feline hypoadrenocorticism are thought to be immune mediated in origin.[5–7] Infiltration of the adrenal glands by lymphoma has also reportedly caused hypoadrenocorticism in two cats.[10]

HISTORY, CLINICAL SIGNS, AND PHYSICAL EXAMINATION

Cats of any age, breed, or sex can develop primary hypoadrenocorticism.[1,3–8] There is no reported sex predilection, and the median age at time of diagnosis is 4 years (age range: 1.5 to 14 years).[1,3,4,6] The typical history of a cat with

*Dr. Redden became an independent contractor for Novartis Animal Health after writing this article.

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Common Findings in Feline Hypoadrenocorticism

Clinical signs	Physical examination	Blood work abnormalities
• Anorexia	• Depression	• Hyponatremia
• Lethargy	• Weakness	• Hypochloremia
• Weight loss	• Dehydration	• Hyperkalemia
• Episodic vomiting	• Hypothermia	• Hyperphosphatemia
• Waxing/waning course	• Shock	• Sodium:potassium ratio <30:1
		• Azotemia
		• ± Hypercalcemia
		• Urine specific gravity <1.030
		• ± Nonregenerative anemia

hypoadrenocorticism includes clinical complaints of lethargy, anorexia, and weight loss.[1–4,6–8] The following are less commonly reported: vomiting, a waxing/waning course of illness, polyuria, polydipsia, previous response to therapy, reluctance to jump, and muscle tremors.[1,3,4,6] Diarrhea and cardiac abnormalities have not been reported as clinical signs in cats with hypoadrenocorticism.[1,3,7] The most common findings during physical examination are nonspecific, including depression, weakness, dehydration, and hypothermia.[1,3,4] One case report described a cat with a presenting complaint of dysphagia caused by muscle weakness.[6]

LABORATORY ABNORMALITIES

Cats with hypoadrenocorticism usually have significant abnormalities on their biochemistry profile and urinalysis. Most have mild to severe prerenal azotemia, hyperphosphatemia, hyponatremia, and an abnormal sodium:potassium ratio.[1–4] The sodium:potassium ratio is often 20:1 or less (30:1 is a normal sodium:potassium ratio).[9] The urine specific gravity is usually not greater than 1.030, despite the presence of dehydration and prerenal azotemia, because loss of renal medullary solutes, especially sodium, results in impaired renal-concentrating ability.[1,3,6] Some cats exhibit hyperkalemia, hypochloremia, and/or hypercalcemia[1]; 13% of cats reported with hypoadrenocorticism were hypercalcemic at presentation.[5] The pathogenesis of hypercalcemia associated with hypoadrenocorticism is unknown but suspected to be multifactorial.[11] The hypercalcemia could be associated with hyperproteinemia secondary to dehydration and hemoconcentration, increased plasma binding of calcium, increased concentration of calcium citrate complexes, and/or increased renal tubular resorption of calcium.[11]

There are no consistent hematologic abnormalities reported in cats with hypoadrenocorticism.[3,4] Lymphocyte and eosinophil counts may be normal, increased, or decreased.[3,4] A normal or increased lymphocyte and eosinophil count is significant because the expected response to stress would be lymphopenia and eosinopenia.[3,5,9,12] Some cats have had mild normocytic, normochromic, nonregenerative anemia.[3,4,8] The box on this page summarizes the most common clinical signs, physical examination findings, and blood work abnormalities reported in cases of feline hypoadrenocorticism.

DIAGNOSTIC TESTING

In most cats with hypoadrenocorticism, thoracic radiographs usually show microcardia and pulmonary hypoperfusion.[1,3,6] Mineralocorticoid deficiency causes sodium loss, resulting in vascular volume contraction and impaired peripheral circulation.[3] Glucocorticoid deficiency reduces vascular responsiveness to catecholamines, contributing to vascular collapse and hypoperfusion on radiographs.[3]

In cats, electrocardiograms rarely show changes due to hyperkalemia.[1,3] Hyperkalemia decreases myocardial contractility and hampers conduction, helping lead to circulatory failure.[3] The typical signs of hyperkalemia on canine electrocardiograms, such as peaking of the T wave, decreased R wave amplitude, and reduced or absent P waves, are not typically seen on electrocardiograms of cats with hypoadrenocorticism.[3,6] Cats with documented hypoadrenocorticism have had milder hyperkalemia compared with dogs with hypoadrenocorticism, helping account for the lack of electrocardiographic changes.[6,7] Some cats with hypoadrenocorticism have had sinus bradycardia or atrial premature contrac-

Cats take longer (i.e., 3 to 5 days) than other species to respond to appropriate treatment of hypoadrenocorticism.

tions on their electrocardiograms, although these signs have not been commonly reported.[1]

Ultrasonography cannot be routinely used to identify small adrenal glands in cats because the right adrenal gland is difficult to see in normal animals.[5] Some recent ultrasonographic studies of the normal feline adrenal gland suggest that ultrasonography may become useful in diagnosing diseases of the adrenal gland in the future as machines become more sensitive and ultrasonographers gain more experience with imaging of the adrenal glands.[13]

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of feline hypoadrenocorticism includes renal disease, gastrointestinal disease, and other cardiovascular, neurologic, muscular, or metabolic diseases that cause weakness.[5,8] If hypercalcemia is present, other causes of hypercalcemia, such as neoplasia and primary hyperparathyroidism, must be considered.[14]

DIAGNOSIS

The main tool used to diagnose hypoadrenocorticism in cats is the ACTH stimulation test.[1,3] There are two main protocols for the ACTH stimulation test in cats. One protocol is to use ACTH gel: The serum or plasma cortisol level is measured before and 60 and 120 minutes after administering ACTH gel intramuscularly (2.2 U/kg).[3,6–8] A second protocol is to inject synthetic ACTH (Cortrosyn, Organon Pharmaceuticals; 125 µg) intravenously or intramuscularly and measure serum or plasma cortisol levels before and 30 and 60 minutes after injection.[3–8] The latter protocol is preferred because synthetic ACTH stimulates the adrenal cortex more consistently and to a greater degree than does ACTH gel.[12] In normal animals, basal cortisol concentrations are usually 0.5 to 5 µg/dl and post-ACTH cortisol concentrations are 4.5 to 13 µg/dl.[7] A low or low-normal baseline serum cortisol level with little or no increase in the serum cortisol level after ACTH administration is diagnostic of hypoadrenocorticism.[1,7,8] In cats with hypoadrenocorticism, pre- and post-ACTH cortisol levels were reported to be less than 2 µg/dl.[7]

Elevation of endogenous plasma ACTH concentration is also useful in confirming primary hypoadrenocorticism in cats.[1,3–5,7] In one study of 10 cats, endogenous plasma ACTH levels were elevated at 500 to 8,000 pg/ml in all cats with hypoadrenocorticism (normal levels: <10 to 125 pg/ml).[3] In cats with primary hypoadrenocorticism, ACTH secretion is increased by a normal pituitary[1]; decreased cortisol production decreases the negative feedback to the pituitary, resulting in increased production of ACTH.[1,5] ACTH rapidly disappears from whole blood, and the diagnostic laboratory should be consulted for information on sample collection and submission if the endogenous plasma ACTH concentration needs to be measured.[3–6]

TREATMENT

Initial

Treatment of hypoadrenocorticism is usually initiated before a definitive diagnosis is obtained because usually results of the ACTH stimulation test are not readily available. A presumptive diagnosis should be based on the history, clinical signs, and sodium:potassium ratio.[9]

Cats with hypoadrenocorticism have vague presenting clinical signs, such as lethargy, anorexia, and weight loss, and almost always have serum electrolyte abnormalities.

Treatment involves restoring the circulating blood volume, correcting serum electrolyte and acid–base imbalances, and providing glucocorticoid and mineralocorticoid support.[3,7] Initial management includes administration of 0.9% saline fluids at a rate of 40 ml/kg/hr IV to correct fluid deficits over the first 2 hours; this is then tapered to a maintenance rate of 60 ml/kg/day.[1,3,4,7] Fluid therapy should be discontinued when azotemia and electrolyte abnormalities resolve and the cat is eating and drinking.[12] Intravenous dexamethasone at a dose of 0.5 to 1 mg/kg q12–24h corrects glucocorticoid deficits and does not interfere with ACTH stimulation testing.[1,3,4,7,8] Mineralocorticoid support is provided by administering intramuscular repository desoxycorticosterone pivalate (DOCP; Percorten-V, Novartis Animal Health; 10 to 12.5 mg/mo) or oral fludrocortisone acetate (Florinef, Bristol-Myers Squibb; 0.10 mg q24h).[1,3,4,7] Most often, the hyperkalemia and mild metabolic acidosis that may be present respond to fluid therapy and other treatment is not needed[3]; fluid

therapy increases renal perfusion, which decreases the serum potassium concentration by dilution and renal potassium excretion.[5]

Once treatment has been initiated, clinical signs usually take longer to resolve in cats than in dogs.[1,3,4,7] Lethargy, weakness, and anorexia often persist for 3 to 5 days.[1,3,4,7] Cats that are diagnosed with hypoadrenocorticism and respond poorly to treatment should be evaluated for lymphoma (via ultrasonography or biopsy) because lymphosarcoma can infiltrate the adrenal glands.[7,12]

Maintenance

Maintenance therapy consists of lifelong mineralocorticoid support with or without glucocorticoid support. Once a cat has been started on intramuscular repository DOCP or oral fludrocortisone acetate, the dose must be adjusted based on evaluations of serum electrolyte concentrations every 1 to 2 weeks until the patient is stable.[4] In most cats, oral fludrocortisone acetate should be administered at a dose of 0.1 mg q24h.[1,3,4] Most cats require intramuscular DOCP every 25 to 30 days.[3,7] Frequent sampling is recommended to determine the time interval for administering DOCP because dogs with hypoadrenocorticism often require DOCP more frequently than every 30 days.[3] Glucocorticoid supplementation is recommended but not required in all cases, and prednisolone (0.5 to 2 mg/cat/day PO) or methylprednisolone (10 mg/cat/mo IM) can be used.[4,6–8] Alternate-day therapy with prednisolone is not required because there is not an intact hypothalmic–pituitary–adrenal axis.[9] The use of monthly intramuscular methylprednisolone may increase the risk of developing diabetes mellitus.[4]

The long-term prognosis for a cat with hypoadrenocorticism is good.[1,5–8] With correct glucocorticoid and mineralocorticoid support, cats with hypoadrenocorticism can have a normal life expectancy.[3] Periodic monitoring of serum electrolytes, blood urea nitrogen, and creatinine is strongly recommended.[7]

CONCLUSION

Feline hypoadrenocorticism is an uncommon disease but should not be overlooked. Relatively few cases have been reported in the literature. The disease has the ability to mimic other disorders, especially renal disease, and cases of feline hypoadrenocorticism have likely gone undiagnosed. Feline hypoadrenocorticism is easy to test for using the ACTH stimulation test, and treatment is rewarding because most cats have an excellent response.

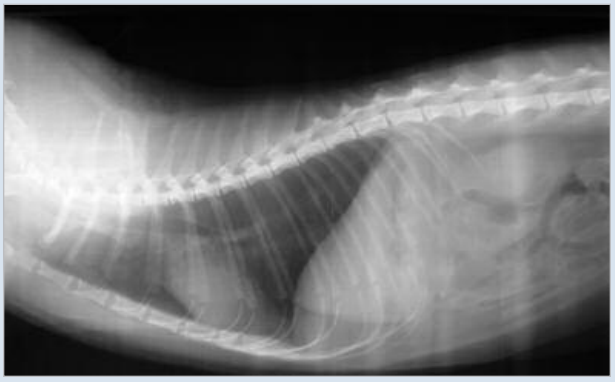
Case Study

A 2½-year-old castrated domestic shorthaired cat presented with a 2-day history of anorexia, weight loss, and lethargy. There had been no vomiting or diarrhea. The cat was housed strictly indoors without other cats and was current on its vaccinations. A review of the medical record revealed that the cat had weighed 12 lb (5.45 kg) the previous year; it currently weighed 10.7 lb (4.86 kg) and had a body condition score of 2.5 of 5 (optimal body condition score: 3 of 5). A physical examination was attempted, but the cat was difficult to handle and became increasingly more fractious as the examination continued. The cat was placed in an anesthesia induction chamber, anesthetized with 5% isoflurane, and maintained with 2.5% isoflurane. A physical examination was performed while the cat was anesthetized, and no major abnormalities were found. Blood was also obtained for a complete blood cell count (CBC), serum biochemistry profile, serum total thyroxine (T_4) level, and viral and serologic testing. The bladder was empty, so urine was not obtained.

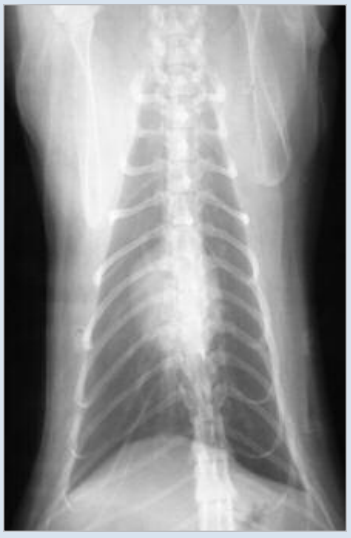
The initial biochemistry profile revealed hyperglycemia, hypercalcemia, hyperkalemia, hyponatremia, hypochloremia, and negative viral and serologic test results (Table 1). The serum glucose level was 230 mg/dl (normal: 64 to 170 mg/dl), the sodium level was 137 mEq/L (normal: 145 to 158 mEq/L), the potassium level was 5.5 mEq/L (normal: 3.5 to 4.6 mEq/L), and the chloride level was 98 mEq/L (normal: 104 to 128 mEq/L). The sodium:potassium ratio was low at 24.9:1 (normal: 30:1). A urinalysis was requested to complete the minimum database. The owners were unsuccessful at collecting urine at home, so the cat was brought to the hospital 10 days later to conduct a urinalysis. The cat's weight had dropped to 9.9 lb (4.5 kg); the cat was mildly dehydrated, and the owner said that it had been vomiting. Urine was obtained, and radiographs of the chest and abdomen were taken. The abdominal radiographs were normal, and the thoracic radiographs revealed microcardia (Figure 1). The cat became very lethargic and easier to handle after the stress of obtaining radiographs. The urinalysis results were within normal limits (Table 1).

This patient's hyperglycemia was considered physiologic as a result of stress. The urinalysis results were normal, with no glucose or ketones, which helped rule out diabetes mellitus. Hypercalcemia was a primary problem, and the differential diagnosis included renal disease, neoplasia or hypercalcemia of malignancy, primary hyperparathyroidism, hypoadrenocorticism, and vitamin D toxicosis. Hypoadrenocorticism was considered because of electrolyte abnormalities (i.e., hyponatremia, hyperkalemia, hypochloremia) but was low on the differential list because of its rarity in cats. The differential diagnosis was discussed with the owner, and further diagnostic testing (e.g., ACTH stimulation test, ultrasonography of the thorax and abdomen) was recommended.

Figure 1. Thoracic radiographs.



Lateral view.



Dorsoventral view.

The blood work was repeated before an ACTH stimulation test was conducted, and the previous abnormal findings were confirmed. Results of the serum biochemistry profile showed that hyponatremia and hypercalcemia were present with mild prerenal azotemia (Table 2). The sodium level was 144.2 mEq/L (normal: 145 to 158 mEq/L), the chloride level was 111 mEq/L (normal: 104 to 128 mEq/L), the calcium level was 15.26 mg/dl (normal: 8.2 to 10.8 mg/dl), and the blood urea nitrogen was 38.2 mg/dl (normal: 14 to 36 mg/dl). The sodium:potassium ratio was low at 29.1:1 (normal: 30:1).

Hypoadrenocorticism and parathyroid disease were the two primary differentials; therefore, an ACTH stimulation test was conducted and a parathyroid (PTH)/ionized calcium sample was submitted to an outside laboratory. The ACTH stimulation test revealed

Case Study *(continued)*

Table 1. Initial Biochemistry Profile

Date	Biochemical Test	Laboratory Value	Reference Range
9/28/01	**CBC**		
	Hemoglobin	15.5 g/dl	9.3–15.9 g/dl
	Hematocrit	43.1%	29%–48%
	Leukocytes	10.8×10^3/µl	$3.5–16 \times 10^3$/µl
	Erythrocytes	9.8×10^6/µl	$5.92–9.93 \times 10^6$/µl
	Mean corpuscular volume	44 femtoliters	37–61 femtoliters
	Mean corpuscular hemoglobin	15.8 pg	11–21 pg
	Mean corpuscular hemoglobin concentration	36 g/dl	30–38 g/dl
	Platelet count	Adequate	Adequate
	Absolute neutrophils	3,996	2,500–8,500
	Absolute bands	0	0–150
	Absolute lymphocytes	6,588	1,200–8,000
	Absolute monocytes	108	0–600
	Absolute eosinophils	108	0–1,000
	Absolute basophils	0	0–150
9/28/01	**Chemistry**		
	Albumin	4 g/dl	2.5–3.9 g/dl
	Alkaline phosphatase	28 U/L	6–102 U/L
	Alanine aminotransferase	63 U/L	10–100 U/L
	Amylase	323 U/L	100–1,200 U/L
	Aspartate aminotransferase	34 U/L	10–100 U/L
	Bilirubin (total)	0.1 mg/dl	0.1–0.4 mg/dl
	Blood urea nitrogen	36 mg/dl	14–36 mg/dl
	Calcium	13.2 mg/dl	8.2–10.8 mg/dl
	Chloride	98 mEq/L	104–128 mEq/L
	Cholesterol	171 mg/dl	5–220 mg/dl
	Creatine phosphokinase	410 U/L	56–529 U/L
	Creatinine	2.4 mg/dl	0.6–2.4 mg/dl
	γ-Glutamyltransferase	<5 U/L	1–10 U/L
	Globulin	3.8 g/dl	2.3–5.3 g/dl
	Glucose	230 mg/dl	64–170 mg/dl
	Lipase	103 U/L	0–205 U/L
	Phosphorus	7.1 mg/dl	2.4–8.2 mg/dl
	Potassium	5.5 mEq/L	3.5–4.6 mEq/L
	Sodium	137 mEq/L	145–158 mEq/L
	Sodium:potassium	24.9:1	≥30:1
	T_4	2 µg/dl	0.8–4 µg/dl
	Triglycerides	85 mg/dl	25–160 mg/dl
	Total protein	7.8 g/dl	5.2–8.8 g/dl

Case Study *(continued)*

Table 1. Initial Biochemistry Profile *(continued)*

9/28/01	**Serology**		
	FeLV antigen/FIV antibody	Negative/negative	Negative/negative
	Feline coronavirus immunofluorescent antibody		
	1:400	Negative	Negative
	1:1,600	Negative	Negative
	Toxoplasma antibody IgG and IgM	Negative	Negative
10/18/01	**Urinalysis**		
	Urine specific gravity	1.045	1.001–1.080
	Protein	Trace	Negative
	pH	6	5.5–7.5
	Urine blood	Negative	Negative
	Glucose	Negative	Negative
	Ketones	Negative	Negative
	Bilirubin	Negative	Negative
	Sediment	Inactive	Inactive

a low baseline serum cortisol level of less than 0.2 µg/dl (normal: 1 to 4 µg/dl) with no response to intravenous administration of Cortrosyn (125 µg) after 45 minutes (0.2 µg/dl; normal: 4.5 to 15 µg/dl), which confirmed hypoadrenocorticism. The PTH value was normal and the ionized calcium value elevated, suggesting parathyroid disease because the normal serum PTH concentration was considered inappropriate for the high total serum calcium and ionized calcium concentrations.

Hypoadrenocorticism was the most pressing concern, so the cat was hospitalized and treated with intravenous 0.9% saline at a rate of 40 ml/kg/hr until the fluid deficits were corrected; the dose was then tapered to 60 ml/kg/day. Dexamethasone (1 mg/kg q24h SC) and DOCP (2.2 mg/kg IM) were administered, and DOCP was repeated every 25 to 30 days. Oral treatment with Florinef was not considered because of the cat's aggressive nature.

When rechecked 2 weeks later, the cat had improved but continued to vomit intermittently. Prednisolone was added at a dose of 2.5 mg q24h for 2 weeks and then reduced to q48h for 2 weeks. Four weeks after diagnosis, the cat returned for another DOCP injection and the owner reported that it was doing well with no vomiting. The cat's weight had increased to 11 lb (5 kg). Periodic follow-up serum biochemical analyses have ranged from slightly abnormal to normal, depending on the length of time between DOCP injections (Table 3). The cat has been treated with 12.5 mg of DOCP q25–35day for 3 years and is relatively normal. Electrolyte imbalances resolve with more frequent (every 25 versus 35 days) DOCP administration. Periodic vomiting resolves with low-dose prednisolone therapy.

DISCUSSION

This case represents a characteristic presentation of hypoadrenocorticism—a rare disease in cats. This case was managed successfully with appropriate treatment. The fractious nature of this cat made it difficult to initially obtain a minimum database. The cat had typical nonspecific clinical signs of feline hypoadrenocorticism, and the serum biochemical analyses revealed the classic electrolyte abnormalities without prerenal azotemia at presentation. This patient also presented with hypercalcemia, which is uncommon in cats with hypoadrenocorticism. Hypercalcemia was investigated but not pursued aggressively because treatment of hypoadrenocorticism was considered more vital at that time. The hypercalcemia resolved after the underlying cause (i.e., hypoadrenocorticism) was treated. Clinical signs of hypoadrenocorticism are often exacerbated by stress; this was evident after restraining the patient for radiography (the cat became lethargic and easier to handle).

The rarity of hypoadrenocorticism placed it lower on

Case Study *(continued)*

Table 2. Follow-Up Biochemistry Profile

Date	Biochemical Test	Laboratory Value	Reference Range
10/10/01	**Chemistry**		
	Albumin	3.4 g/dl	2.5–3.9 g/dl
	Blood urea nitrogen	38.2 mg/dl	14–36 mg/dl
	Calcium	15.26 mg/dl	8.2–10.8 mg/dl
	Chloride	111 mEq/L	104–128 mEq/L
	Creatinine	2.47 mg/dl	0.6–2.4 mg/dl
	Globulin	4.7 g/dl	2.3–5.3 g/dl
	Packed cell volume	49%	29%–48%
	Potassium	4.95 mEq/L	3.5–4.6 mEq/L
	Sodium	144.2 mEq/L	145–158 mEq/L
	Sodium:potassium	29.1:1	≥30:1
	Total protein	8.1 g/dl	5.2–8.8 g/dl
10/10/01	**Urinalysis**		
	Urine specific gravity	1.045	1.001–1.080
	Protein	Trace	Negative
	pH	6.5	5.5–7.5
	Urine blood	Negative	Negative
	Glucose	Negative	Negative
	Ketones	Negative	Negative
	Bilirubin	Negative	Negative
	Sediment	Inactive	Inactive
10/11/01	Cortisol (resting)	<0.2 µg/dl	1–4 µg/dl
	Cortisol (after ACTH)	<0.2 µg/dl	4.5–15 µg/dl
10/12/01	Parathyroid hormone (intact)	13.6 pg/ml	0–40 pg/ml
	Ionized calcium	2.07 mmol/L	1.16–1.34 mmol/L
	Calcium	15 mg/dl	8.2–10.8 mg/dl

Table 3. Follow-up Serum Biochemical Analyses

	11/12/01	7/16/02	4/21/03	6/23/04	7/27/04	Reference Range
Sodium (mEq/L)	160	146	157	139	149	145–158
Potassium (mEq/L)	5.1	5.0	4.3	5.1	4.7	3.5–4.6
Calcium (mg/dl)	10.2	—	—	—	8.8	8.2–10.8

Case Study *(continued)*

the differential list than it should have been considering the history, blood work, and radiographs. A plasma ACTH concentration could have been measured before replacement therapy to confirm a diagnosis of primary hypoadrenocorticism but was not needed to decide the treatment and was an unnecessary expense for the client.

Blood work, specifically electrolyte values every 2 to 4 weeks, should have initially been performed more frequently to adjust the time interval and dose of DOCP injections. Owner financial concerns forced the time interval to be adjusted based on clinical signs. This worked adequately for this patient, but basing the time interval for injections on serum electrolyte concentrations would have been more accurate.

This cat responded appropriately to mineralocorticoid support with monthly injections of DOCP but needed periodic glucocorticoid support with prednisolone to control signs. Three years after diagnosis, the cat continues to be well managed and has an excellent long-term prognosis.

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1. Which statement regarding the pathogenesis of feline hypoadrenocorticism is correct?

a. The pathogenesis remains unknown.
b. Secondary hypoadrenocorticism has been reported in 22 cats.
c. Infiltration of the adrenal gland by lymphoma can cause hypoadrenocorticism in cats.
d. a and c
e. a and b

2. A cat with hypoadrenocorticism could have the following abnormality(ies) on serum biochemical analyses:

a. prerenal azotemia, hyperkalemia, and hyponatremia.
b. a sodium:potassium ratio <30:1.
c. hypercalcemia.
d. a and b
e. a, b, and c

3. A cat with hypoadrenocorticism may have the following serum electrolyte abnormalities:

a. hypernatremia, hyperkalemia, hypochloremia, hypercalcemia, and hypophosphatemia.
b. hypernatremia, hypokalemia, hyperchloremia, hypercalcemia, and hyperphosphatemia.
c. hyponatremia, hypokalemia, hypochloremia, hypercalcemia, and hypophosphatemia.
d. hyponatremia, hyperkalemia, hypochloremia, hypercalcemia, and hyperphosphatemia.
e. none of the above

4. Which diagnostic test(s) for feline hypoadrenocorticism is the best?
a. ACTH stimulation test
b. low-dose dexamethasone suppression test
c. endogenous plasma ACTH concentration
d. electrocardiography and radiography
e. serum biochemistry profile and CBC

5. Which is the most likely rule out for a disease that mimics feline hyperadrenocorticism?
a. diabetes mellitus
b. lymphoma
c. pancreatitis
d. renal disease
e. primary hyperparathyroidism

6. Treatment of feline hypoadrenocorticism
a. should not begin until results of the ACTH stimulation test have been evaluated.
b. involves intravenous saline, dexamethasone, and mineralocorticoid support.
c. involves correction of hyperkalemia and metabolic acidosis with insulin therapy and sodium bicarbonate administration.
d. does not require lifelong therapy.
e. quickly resolves clinical signs.

7. Cats with primary hypoadrenocorticism have _____ endogenous plasma ACTH production.
a. increased
b. decreased
c. normal
d. none of the above
e. all of the above

8. Cats with hypoadrenocorticism may present with
a. diarrhea and vomiting.
b. weight loss and anorexia.
c. lethargy.
d. all of the above
e. b and c

9. Which statement regarding the diagnosis of feline hypoadrenocorticism is correct?
a. Radiographs usually show microcardia and pulmonary hypoperfusion.
b. Ultrasonography can readily identify small adrenal glands.
c. Electrocardiograms commonly show changes such as reduced or absent P waves and decreased P wave amplitude because of hyperkalemia.
d. a and b
e. b and c

10. Maintenance treatment of feline hypoadrenocorticism includes
a. intramuscular repository DOCP every 25 to 30 days.
b. oral fludrocortisone acetate once daily.
c. oral prednisolone (0.5 to 2 mg/cat sid), if needed.
d. periodic monitoring of serum electrolyte concentrations.
e. all of the above