Feline hypoadrenocorticism: Emerging insights into clinical features, diagnosis, and treatment

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Hypoadrenokortizismus bei Katzen: Neue Erkenntnisse zu klinischen Merkmalen, Diagnose und Behandlung

Der Hypoadrenokortizismus ist eine seltene Endokrinopathie der Katzen, an die bei unspezifischen, intermittierenden oder schubweise auftretenden klinischen Symptomen gedacht werden sollte. Bei chronischen gastrointestinalen Beschwerden, mangelnder Gewichtszunahme oder wiederkehrenden Episoden eines hypovolämischen Schocks sollte an einen Hypoadrenokortizismus als Verdachtsdiagnose gedacht werden. Britisch-Kurzhaar-Katzen könnten überrepräsentiert sein, weshalb bei dieser Rasse bei entsprechenden Symptomen besondere Aufmerksamkeit geboten ist.

Die definitive Diagnose erfordert einen ACTH-Stimulationstest, der vor Beginn einer Glukokortikoidtherapie durchgeführt werden muss, um diagnostische Verfälschungen zu vermeiden. Bei klinisch stabilen Katzen kann die Messung des basalen Cortisolspiegels als initialer Screening-Test herangezogen werden. Da jedoch bislang keine katzenspezifischen Schwellenwerte validiert wurden, sollte der häufig verwendete Grenzwert zum Ausschluss eines Hypoadrenokortizismus von >2 µg/dL (>55 nmol/L) mit Vorsicht interpretiert werden, da er möglicherweise weniger zuverlässig ist als beim Hund.

Die Langzeittherapie umfasst eine lebenslange Glukokortikoidsubstitution, typischerweise mit oral verabreichtem Prednisolon. Katzen benötigen im Vergleich zu Hunden in der Regel höhere Erhaltungsdosen, um eine klinische Stabilität zu erreichen. Der Mineralokortikoidmangel wird durch eine monatliche subkutane Injektion des Depotpräparats Desoxycorticosteron (Zycortal®) in einer Anfangsdosis von 2,2 mg/kg behandelt; diese Startdosis liegt deutlich über der für den Hund empfohlenen Dosis. Alternativ könnte eine tägliche orale Gabe von Fludrocortisonacetat (Florinef® oder Astonin®H) in einer Dosis von 0,01–0,02 mg/kg/Tag erfolgen. Die regelmässige Kontrolle der Natrium- und Kaliumwerte ist essenziell, um die Mineralokortikoiddosis individuell anzupassen und die Therapie zu optimieren.

Da betroffene Katzen keine adäquate Stressreaktion entwickeln können, ist während Stresssituationen oder begleitender Er-

Summary

Hypoadrenocorticism in cats is a rare endocrine disorder that should be considered in patients presenting with vague, intermittent, or waxing and waning clinical signs. Chronic gastrointestinal signs, poor growth, or episodes of hypovolemic shock should raise clinical suspicion. British Shorthair cats may be overrepresented among affected cats, and clinicians should be particularly alert when this breed presents with compatible signs.

Definitive diagnosis requires an ACTH stimulation test, which should be performed prior to the initiation of any glucocorticoid treatment to avoid diagnostic interference. In clinically stable cats, baseline cortisol measurement may be used as an initial screening tool. However, as no feline-specific thresholds have been validated, the commonly used cut-off of >2 µg/dL (>55 nmol/L) to rule out hypoadrenocorticism should be interpreted with caution, as it may be less reliable than in dogs.

Long-term management involves lifelong glucocorticoid replacement, typically using oral prednisolone. Cats generally require higher maintenance doses than dogs to maintain clinical stability. Mineralocorticoid deficiency is managed with subcutaneous administration of an extended-release desoxycorticosterone preparation (Zycortal®) given monthly at a starting dose of 2,2 mg/kg – substantially higher than canine doses. As an alternative, daily oral fludrocortisone acetate (Florinef® or Astonin®H) could be used at 0,01–0,02 mg/kg/day in selected cases. Regular monitoring of sodium and potassium levels is essential to adjust mineralocorticoid dosages and optimize treatment.

Because affected cats cannot mount an adequate stress response, additional glucocorticoid supplementation («glucocorticoid boost») is required during stressful situations or concurrent illness. Owners must be educated about the signs of adrenal crisis, which can be more subtle and difficult to recognize in cats compared to dogs. Emphasis should be placed on consistent medication administration and routine veterinary monitoring to ensure long-term disease control and quality of life.

Keywords: adrenal gland, ACTH-stimulation, cortisol, glucocorticoid, hyperkalemia, mineralocorticoid

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krankungen eine zusätzliche Glukokortikoidgabe («Glukokortikoid-Boost») erforderlich. Besitzer sollten umfassend über die Anzeichen einer Nebennieren-Krise aufgeklärt werden, die bei Katzen subtiler und schwieriger zu erkennen sein kann als bei Hunden. Eine konsequente Medikamentengabe und regelmässige tierärztliche Kontrollen sind entscheidend für eine langfristige Krankheitskontrolle und eine gute Lebensqualität.

Schlüsselwörter: ACTH-Stimulation, Cortisol, Glukokortikoid, Hyperkaliämie, Mineralokortikoid, Nebennieren

Introduction

While hypoadrenocorticism is rare yet relatively well characterized in humans and dogs, it remains exceedingly uncommon and underreported in cats, with fewer than 100 published cases since 1983.[1-4,8,9,13,14,16,20,22,23,28,29,32,34,35,37,38,40-42,44-48,50,52] The current veterinary literature consists mainly of case reports and a few larger case series. These recent series have provided new insights into the clinical features, diagnostic challenges, and management of feline hypoadrenocorticism, helping to refine understanding of this rare endocrine disorder.[17,39]

Given this limited yet gradually expanding body of evidence, the aim of the present review was to synthesize current knowledge on feline hypoadrenocorticism by integrating both newly published and earlier case reports. In this context, this review provides an updated and comprehensive summary of the current understanding of the disease, including its etiology, clinical presentation, laboratory findings, diagnostic approaches, therapeutic management, and prognosis.

Etiology/Pathophysiology

The adrenal cortex comprises three distinct zones, each with unique functions and morphological characteristics. The *zona glomerulosa*, the outermost layer, synthesizes mineralocorticoids (mainly aldosterone), while the middle and inner layers, the *zona fasciculata* and *zona reticularis,* produce glucocorticoids (primarily cortisol) and androgens. Aldosterone secretion is mainly regulated by the renin-angiotensin system and potassium levels, whereas cortisol production depends on hypothalamic-pituitary-adrenal axis stimulation via adrenocorticotropic hormone (ACTH). A comprehensive understanding of the various forms of hypoadrenocorticism necessitates an understanding of the physiological regulation of adrenal cortex hormone production summarized in Figure 1.

Hypoadrenocorticism is classified as primary, due to adrenal cortical dysfunction, or secondary, from inadequate pituitary ACTH production. A tertiary form caused by deficient hypothalamic corticotropin-releasing hormone (CRH) is hypothesized but undocumented in veterinary medicine. Primary hypoadrenocorticism usually results from progressive immune-mediated cortical destruction and atrophy (Addison's disease). In rare cases, only specific adrenal layers are affected, leading to isolated glucocorticoid or mineralocorticoid deficiency. The former is referred to as eunatremic, eukalemic primary hypoadrenocorticism, whereas the latter, which affects the *zona glomerulosa*, results in isolated hyperreninemic hypoaldosteronism.

Although hypoadrenocorticism remains exceedingly uncommon in cats, reports suggest similar pathological mechanisms. Lymphocytic infiltration of the adrenal cortex has been de-

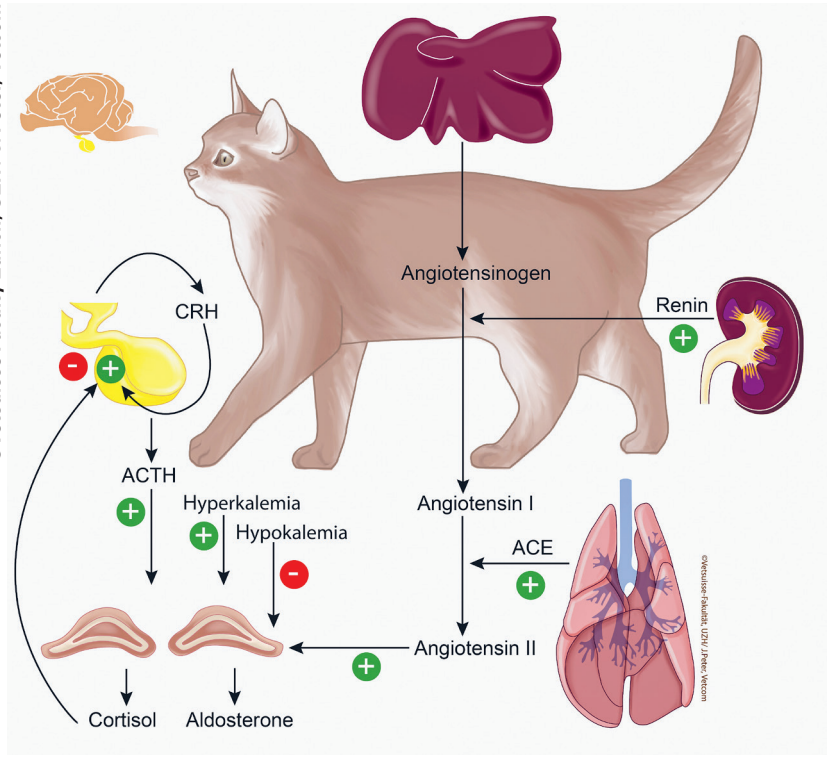

Figure 1: Schematic illustration of the cortisol and aldosterone production from the adrenal glands.

scribed in post-mortem histological examinations, supporting an immune-mediated etiology.[38] Adrenal destruction may also occur secondary to lymphoma (4,9 % of cats in one large series), visible as adrenal masses or enlargement on imaging, highlighting the need to screen for lymphoma.[32,39,40] Other feline causes include trauma-induced adrenal insufficiency (two cases), congenital disease (two kittens), and acute adrenal necrosis with neutrophilic inflammation.[4,8,9,16,28] Additional, less common mechanisms include granulomatous infiltration, hemorrhagic infarction, amyloidosis, or iatrogenic necrosis from mitotane or trilostane.[28]

Secondary hypoadrenocorticism, far rarer than primary disease, stems from ACTH deficiency and primarily causes isolated cortisol deficiency because only the middle and inner adrenal zones atrophy. Causes include pituitary trauma, tumors, lymphocytic hypophysitis, infection, vascular events, congenital defects, hypophysectomy, or, most commonly, iatrogenic suppression from long-term corticosteroid or progestogen treatment with abrupt withdrawal.[30,41,51] In feline patients, treatment with methylprednisolone acetate (20 mg/week, administered subcutaneously for 1–4 weeks) has been shown to produce low to low-normal resting serum cortisol concentrations and an inadequate response to ACTH stimulation. Similarly, oral administration of prednisolone (≥ 2 mg/kg once daily for at least 14 days) suppresses adrenal function, although serum cortisol levels typically normalize within 30 days after discontinuation of treatment. Severe, transient adrenocortical suppression has also been observed following the administration of the synthetic progestogen megestrol acetate.

Signalment

Affected cats of published cases have a median age of approximately 5 years, with cases ranging from young to geriatric cats. Neutered males are most commonly affected, followed by neutered females, whereas intact cats of both sexes are only rarely described. Domestic Shorthair and Domestic Longhair cats are the most frequently reported breeds, likely reflecting their general population prevalence. In some studies, British Shorthair cats appear overrepresented, suggesting a possible genetic predisposition.[39,40] A comprehensive summary of the signalment characteristics is provided in Table 1.

Clinical features

Hypoadrenocorticism in cats can manifest with clinical signs that vary from subtle and chronic to sudden and life-threatening.[1-4,8,9,13,14,16,20,22,23,28,29,32,34,35,37,38,40-42,44-48,50,52] Many affected cats present with lethargy, poor appetite or anorexia, and weight loss. Weakness is also frequently noted and may progress to episodes of collapse. Gastrointestinal disturbances include vomiting and, less commonly, diarrhea or constipation; these signs sometimes improve transiently with fluid therapy or corticosteroid administration. Some cats exhibit neurological abnormalities, such as tremors, seizures, or ataxia, reflecting the potential impact of electrolyte imbalance or hypoglycemia on neuromuscular function.[27] Occasionally, regurgitation or dysphagia is observed when severe muscle weakness affects swallowing reflexes. While polyuria and polydipsia can occur, some cats show diminished thirst or even adipsia. The onset of clinical signs can range from a few days to several months, and cats

Table 1: Signalment of cats with hypoadrenocorticism

Signalment	Median (range)	Number of cats/ total	Percent of total
Age			
Years	5,7 (0,2–13,8)[17] 4 (0,7–14)[39]		
Sex (n=79)			
Neutered Males		42/79	53,16 %*
Neutered Females		30/79	37,97 %*
Entire Females		6/79	7,59 %*
Entire Males		1/79	1,27 %*
Breed (n=80)			
Domestic Shorthair/Longhair		54 /80	67,5 %*
British Shorthair		13/ 80	16,25 %*
Siamese		4 /80	5 %
Other Breeds (e.g. Bengal, Tonkinese)		9/ 80	11,25 %

* Numbers compiled from the two largest publications[17,39]

may experience waxing and waning symptoms before the underlying condition is identified. Typical clinical signs in the history of cats with hypoadrenocorticism that have been described are listed in Table 2.

Physical examination of cats with hypoadrenocorticism frequently reveals dehydration and hypothermia, often accompanied by a weak pulse or prolonged capillary refill time, along with general weakness or marked lethargy (Figure 2).

Some individuals present with a faint cardiac murmur, bradycardia, or even collapse if they are presented in an adrenal crisis. When neurologic deficits are present, they can manifest as abnormal postural reactions, tremors, or seizures, though these appear less commonly. Rarely, a painful abdomen or tachypnea has been described. Clinical signs and physical examination findings reported in cats are summarized in Table 3.

Table 2: Clinical signs reported in the history of cats with hypoadrenocorticism

Clinical signs	Number of affected cats*	Total number of cats*	Percent of total*
Lethargy	61	89	68,5
Anorexia/hyporexia	59	89	66,3
Weakness	34	89	38,2
Weight loss	34	89	38,2
Vomiting	30	89	33,7
Diarrhea	11	41	26,8
Collapse	10	41	24,4
Nausea	9	41	22,0
Polyuria/polydipsia	19	89	21,3
Constipation	13	89	14,6
Seizures/tremors	6	41	14,6
Hypodipsia/adipsia	8	89	9,0
Ataxia	3	48	6,3
Hypothermia	3	48	6,3
Dysphagia/difficulty swallowing	5	89	5,6
Weight gain	2	41	4,9
Previous response to steroid therapy	2	48	4,2
Waxing and waning clinical signs	2	48	4,2
Empty swallowing	2	48	4,2
Polyphagia	3	89	3,4
Hematochezia	1	41	2,4
Hair loss	1	48	2,1
Periuria	1	48	2,1
Hindlimb weakness	1	48	2,1
Difficulty jumping/climbing	1	48	2,1
Clinginess	1	48	2,1
Bilateral mydriasis	1	48	2,1

* Approximate numbers compiled from the two largest publications[17,39]

Hypoadrenocorticism is already a rare endocrinopathy in cats, and eunatremic, eukalemic presentations are even less frequently documented, making it difficult to draw clear distinctions between these non-classical cases and those with classic electrolyte derangements. In one larger retrospective cohort, these eunatremic, eukalemic individuals frequently presented with vomiting but showed fewer characteristic signs on physical examination, such as marked weakness, hypothermia, or dehydration.[39]

Clinicopathologic Findings

Hematology

Various hematological abnormalities have been described (see Table 4).[1-29] Anemia (normocytic, normochromic, non-regenerative) and neutrophilia are among the more frequent hematological abnormalities; notably, cats are less likely than dogs to lack a stress leukogram and even more rarely exhibit lymphocytosis and eosinophilia; in some cases, even a stress leukogram has been reported despite cortisol deficiency.

Biochemistry

Routine laboratory evaluations typically demonstrate findings consistent with mineralocorticoid deficiency, notably hyponatremia and hyperkalemia (see Table 4).[1-29] Although many cats exhibit decreased sodium-to-potassium ratios (<27:1), normal ratios are sometimes encountered, therefore it is recommended to rely on sodium and potassium concentrations separately, instead of the ratios.

Azotemia and hyperphosphatemia are also common and are presumed to result from volume contraction and decreased renal perfusion due to dehydration in cats presenting with vomiting and diarrhea. It is important to note that the azotemia associated with hypoadrenocorticism typically represents a pre-renal azotemia.

Additional clinicopathological findings can include hypercalcemia, hypocholesterolemia, hypoalbuminemia, and, in some cases, elevated creatine kinase activity. Notably, in the most recent review, approximately one-third of affected cats were reported to be hypercalcemic.[39,45] In cats with hypoadrenocorticism, hypoglycemia, and, unlike in dogs, also hyperglycemia have been described. In a subset of affected cats, cobalamin deficiency and low feline trypsin-like immunoreactivity (fTLI) have been reported.[39] Exocrine pancreatic insufficiency or chronic enteropathies may coexist, particularly among cats presenting with marked weight loss or persistent gastrointestinal signs.[39] However, suspected chronic enteropathy in the reported cases has not been confirmed by intestinal biopsies.

Urinalysis

Ideally, a urine sample should be collected prior to initiating fluid therapy in cats with hypoadrenocorticism. Assessment of urine concentrating ability often reveals lower specific

gravity than expected in dehydration and hypovolemia (values below 1,030 or 1,035), suggestive of medullary washout secondary to ongoing sodium loss.[1-4,8,9,13,14,16,20,22,23,28,29,32,34,35,37,38,40-42,44-48,50,52] Therefore, differentiating hypoadrenocorticism from kidney disease, particularly acute kidney injury, can be challenging in certain cases.

Diagnosis

ACTH stimulation Test

The ACTH stimulation test is the test of choice for confirming the diagnosis of hypoadrenocorticism. In unstable patients, it is recommended to proceed always directly to this test without waiting for the baseline cortisol results. Whenever possible, prior administration of glucocorticoids should be avoided, as it can make test interpretation impossible (see below).

Because hypoadrenocorticism is rare in cats, some authors advise performing an ACTH stimulation test in any clinically suspect case; however, in stable patients with a low pre-test likelihood, a single basal cortisol measurement can be used as an initial screening test (see below).

Test performance

The evidence base for the standardized ACTH stimulation testing protocol in cats remains limited.[25] Two dosages are described in the literature: a fixed 125 µg dose per cat and a weight-based dose of 5 µg/kg, the latter extrapolated from canine practice. Although no studies have evaluated lower ACTH dosages in this species, the weight-based dose is widely used in clinical practice. A synthetic form of ACTH, such as cosyntropin or tetracosactrin, is administered intravenously at this supraphysiologic dose.

Compared to dogs, the cortisol response to ACTH in cats appears to be more variable, making the timing of the peak response less predictable. As a result, two post-ACTH sampling time points are often recommended. Commonly used time points in addition to baseline (0 minutes), include 30, 60, 90, or 120 minutes post-ACTH administration. The authors typically utilize 0, 30, and 60 minutes for sampling. However, in cats that are very small, anemic, or difficult to sample, a simplified protocol using only a single 60-minute post-ACTH sample may be a reasonable alternative.

Interpretation

In cats with normal adrenal gland function, the administration of ACTH leads to a significant increase in cortisol concentration, typically surpassing 5 µg/dL (138 nmol/L), although this reference value is extrapolated from canine data.[19,36] In one larger case series, post-ACTH stimulation cortisol concentrations were either undetectably low or had a median concentration of 0,9 µg/dL (24,8 nmol/L), with a range of 0,1–1,8 µg/dL (2,8–50 nmol/L). [44] Therefore, post-stimulation cortisol concentrations below 2 µg/dL (55 nmol/L) can be regarded as confirmatory, consistent with findings in dogs. Values exceeding 5 µg/dL (138 nmol/L) indicate an adequate adrenal reserve, whereas concentrations between 2 and 5 µg/dL (55–138 nmol/L) are considered borderline and fall within the gray zone. Various factors can contribute to an inadequate response to ACTH stimulation besides hypoadrenocorticism. The administration of gluco-

Table 3: Physical examination findings reported in cats with hypoadrenocorticism

Physical examination findings	Number of affected cats*	Total number of cats*	Percent of total*
Weakness	26	40	65,0
Dehydration	50	88	56,8
Hypothermia	35	84	41,7
Neurological abnormalities	9	40	22,5
Weak pulse	8	48	16,7
Tachypnea	11	82	13,4
Abdominal pain	10	88	11,4
Heart murmur	10	88	11,4
Collapse	9	88	10,2
Slow capillary refill time	5	48	10,4
Lateral recumbency	4	48	8,3
Bradycardia	6	88	6,8
Seizures	1	48	2,1

* Numbers compiled from the two largest publications[17,39]

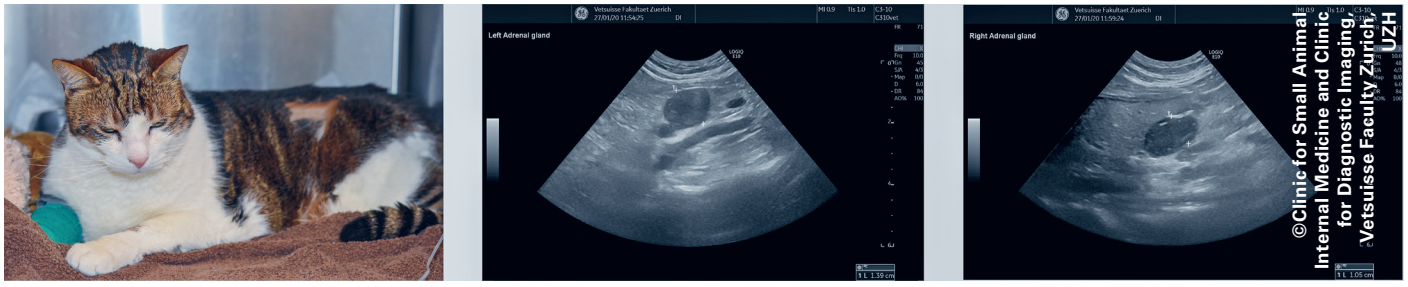

Figure 2: A 12-year-old European Shorthair cat with hypoadrenocorticism secondary to metastatic round cell neoplasia involving both adrenal glands. Ultrasound images of the left and right adrenal glands show marked bilateral enlargement with a rounded shape, surrounded by hyperechoic tissue.

corticoids (topically or systemically) or progestins prior to the test is a significant factor.[10] Depending on the glucocorticoid product and duration of use, post-stimulation cortisol concentrations of 2 to 5 µg/dl (55 to 138 nmol/l) or even values below the detection limit of the cortisol assay due to suppression of the hypothalamic-pituitary-adrenal axis are possible. Additionally, synthetic glucocorticoids (e.g., prednisolone, methylprednisolone) given on the same day as the test can directly interfere with cortisol measurement and lead to falsely elevated cortisol values. However, this interference does not occur when using dexamethasone. Even so, because interpretation of the ACTH stimulation test is more challenging in cats than in dogs, any glucocorticoid administration before testing (including dexamethasone) should be avoided whenever clinically feasible. If glucocorticoid support is unavoidable in an emergency, dexamethasone should be used, as it does not interfere with cortisol measurement. Dexamethasone, like other glucocorticoids, inhibits endogenous cortisol production directly, but this effect typically takes several hours to manifest. To avoid artificially decreased post-ACTH cortisol concentrations, it is crucial to ensure that the ACTH stimulation test is conducted within a few hours after administering dexamethasone. By doing this, correct interpretation and exclusion of hypoadrenocorticism should still be possible after a single dose of dexamethasone.

Table 4: Abnormal laboratory values in cats with hypoadrenocorticism

Laboratory abnormality	Number of affected cats*	Total number of cats*	Percent of total*
Hematology			
Absence of stress leukogram	29	89	32,6
Anemia	19	89	21,3
Lymphocytosis	14	89	15,7
Neutropenia	7	41	17,1
Neutrophilia	7	48	14,6
Eosinophilia	4	41	9,8
Serum biochemistry			
Azotemia	60	89	67,4
Hyponatremia	59	89	66,3
Hyperkalemia	54	89	60,7
Hypercalcemia (ionized calcium only)	8	14	57,1
Hypochloremia	39	86	45,3
Increased AST	11	27	40,7
Hyperphosphatemia	33	88	37,5
Increased CK	27	75	36,0
Hyperglycemia	13	37	35,1
Hypercalcemia (total or ionized, or both)	13	40	32,5
Hypoalbuminemia	12	40	30,0
Hypoglycemia	10	39	25,6
Hypercalcemia (total calcium only)	10	40	25,0
Na:K ratio <27	12	48	25,0
Increased ALT	21	89	23,6
Na:K ratio normal	10	48	20,8
Hypoglobulinemia	6	30	20,0
Hyperbilirubinemia	7	37	18,9
Increased AP	7	39	17,9

* Numbers compiled from the two largest publications[17,39]

The ACTH stimulation test alone cannot distinguish between different forms of adrenal insufficiency in animals. These include primary hypoadrenocorticism, eunatremic, eukalemic primary hypoadrenocorticism, secondary adrenal insufficiency resulting from chronic pituitary dysfunction, or secondary adrenal insufficiency due to recently discontinued long-term glucocorticoid administration. Therefore, in cases in which electrolyte concentrations are within the reference range, it is crucial to thoroughly exclude any prior administration of glucocorticoids. Additionally, measuring endogenous ACTH concentration can be helpful in differentiating between these various forms (see below).

Baseline serum cortisol

In clinically stable cats with low suspicion of hypoadrenocorticism, a single baseline serum cortisol can serve as an initial screening test. Extrapolating from canine data, levels > 2 µg/dL (> 55 nmol/L) typically exclude the disease, but this cut-off lacks feline-specific validation and should be interpreted cautiously.[6]

Endogenous ACTH

In cats with primary hypoadrenocorticism, plasma ACTH concentrations are typically markedly elevated, often exceeding the established reference interval.[39,44] In contrast, cats with secondary hypoadrenocorticism – whether pituitary-dependent or iatrogenic due to exogenous glucocorticoid administration – generally exhibit subnormal or undetectable ACTH levels. A chemiluminescent immunometric assay (Immulite 2000 Siemens Healthineers, Erlangen, Germany) has been validated for feline plasma, with a very wide reference interval of 32–370 pg/mL.[49] In that study, cats with primary hypoadrenocorticism showed ACTH concentrations well above this range, whereas healthy cats and those with non-endocrine illnesses did not.[49] Determination of ACTH can be beneficial in cases in which ACTH stimulation test results are in the gray zone and in which serum electrolytes are within the reference range. In these cases, increased ACTH concentrations can indicate primary, eunatremic and eukalemic hypoadrenocorticism.

Sample processing is important as endogenous ACTH is unstable and improper handling can lead to erroneously low results. Chilled EDTA plasma should be collected and promptly separated and frozen within 30 minutes of collection to ensure accuracy. Furthermore, it is essential to avoid contact with glass, and to use plastic tubes instead, as the ACTH can adhere to glass. Once separated, the plasma can be stored at -20°C for an extended period.

Specific sample collection and handling procedures may vary depending on the assay utilized. Therefore, it is advisable to consult the laboratory performing the analysis for their recommended guidelines regarding sample handling. Additionally, it is crucial to inquire whether the assay has been validated for use in cats and if reference ranges are available.

Cortisol-to-ACTH ratio

The cortisol-to-ACTH ratio (CAR) has been proposed as an alternative to the ACTH stimulation test in dogs where synthetic ACTH is unavailable or costly.[5,24] Dogs with primary hypoadrenocorticism exhibit low cortisol and high endogenous ACTH concentrations, resulting in a markedly decreased CAR. In cats, due to the rarity of the disease, CAR has not been systematically evaluated, and interpretation guidelines are lacking. Only one case report described diagnosing hypoadrenocorticism in a cat based on a very low CAR when ACTH stimulation testing was not possible.

Aldosterone

Measurement of aldosterone in cats with suspected primary hypoaldosteronism is rarely performed. Reported cases showed concentrations below the reference interval or detection limit, both before and after ACTH stimulation.[39] Some cats with eunatremic, eukalemic hypoadrenocorticism also had undetectable aldosterone levels, making it unreliable for distinguishing primary from secondary disease.[39] Thus, aldosterone measurement has limited diagnostic value.

Plasma renin activity and aldosterone to renin ratio

Aldosterone deficiency reduces sodium reabsorption, causing hypovolemia and elevated plasma renin activity. In primary hypoadrenocorticism, this renin increase accompanies low aldosterone levels. Due to the rarity of the disease, renin activity has not been systematically studied in cats.[21]

Diagnostic imaging: Ultrasonography of the adrenal glands

Abdominal imaging and ultrasonographic adrenal measurements are important components in the diagnostic evaluation of suspected hypoadrenocorticism in cats.[18,53] However, findings must be interpreted in conjunction with clinical signs and endocrine testing.[11,12] Since adrenal lymphoma has been reported as a more common cause of hypoadrenocorticism in cats than in dogs, enlarged adrenal glands or even mass lesions may be seen (Figure 2). Most cats with hypoadrenocorticism, however, have small adrenal glands with widths between 1 mm and 3 mm. Measurements ≤ 2 mm on the left or ≤ 2,7 mm on the right – or complete non-visualization of the adrenals – should heighten suspicion.[38,39,44] It is important to note that body weight has been shown to influence adrenal size in cats and that small cats with a body weight ≤ 4 kg can have adrenal gland measurements as small as 2,2 mm.[33] Chronic oral corticosteroid administration can potentially result in adrenal atrophy visible on ultrasound. However, in contrast to dogs, the atrophy in cats seems to be less pronounced.[15] In one study, only cats receiving anti-inflammatory doses for more than one month showed a mean decrease, which was only mild (about 0,8 mm), and no practical size cut-off could distinguish treated from untreated animals.

Treatment and Follow-up

The treatment of hypoadrenocorticism can be divided into two phases: treatment during the adrenal crisis and the long-term treatment.

Treatment during the adrenal crisis

Acute adrenal crisis represents a life-threatening emergency. Therefore, immediate initiation of treatment is required for every patient with strong clinical evidence of adrenal insufficiency. The initial emergency treatment aims to restore fluid volume, correct electrolyte imbalances, and as a next step, administer rapid-acting glucocorticoids. Simultaneously with emergency care, further diagnostic evaluation should be conducted and an ACTH stimulation test performed. Whenever possible, it is important to perform the ACTH-stimulation test and confirm the diagnosis before starting glucocorticoid treatment, as glucocorticoids can complicate the diagnostic work-up (see above). While long-term treatment for cats with primary hypoadrenocorticism involves both glucocorticoid and mineralocorticoid treatment, mineralocorticoid treatment is not necessary during the acute phase of treatment but should only be started after the cat has been stabilized.

Fluid therapy

Fluid therapy in feline hypoadrenocorticism should primarily aim to correct hypovolemia, dehydration, and electrolyte imbalances. In cases of hypovolemic shock, isotonic crystalloids with low potassium concentrations (e.g. Plasma-Lyte A, Normosol-R, or Ringer's solution) are recommended as first-line fluids. These solutions support renal perfusion, help correct metabolic acidosis, and avoid the acidifying effects associated with 0,9 % saline.

Particular caution is required in patients with severe hyponatremia, where sodium correction must proceed slowly to prevent osmotic demyelination and associated neurological complications.[31] Likewise, hyperkalemia often improves with adequate fluid therapy alone, though severe cases may require emergency interventions to stabilize cardiac function and lower serum potassium.

Because the detailed management of shock, fluid resuscitation, and electrolyte correction in cats is beyond the scope of this review, readers are referred to established veterinary

internal medicine and critical care literature for comprehensive treatment protocols and monitoring guidelines.

Glucocorticoid replacement

Glucocorticoid treatment should ideally be delayed until diagnostic tests for hypoadrenocorticism are completed. Delaying the initiation of glucocorticoid treatment is usually not problematic, as long as adequate intravenous fluid treatment has been implemented. If the patient is hemodynamically unstable, intravenous (IV) dexamethasone (0,25–0,5 mg/kg) can be given concurrently with the ACTH stimulation test, as it does not cross-react with most cortisol assays. After completing the test, replacement therapy can be initiated using rapid-acting glucocorticoids (e.g., prednisolone or hydrocortisone sodium succinate).[43]

If only dexamethasone is available for parenteral administration, 0,05–0,1 mg/kg every 12–24 hours can be administered until transitioning to oral prednisolone. Our clinic typically uses prednisolone sodium succinate starting at 2 mg/kg IV, followed by 1 mg/kg IV every 6 hours for 24 hours. The dose is then reduced to 0,5 mg/kg twice daily until the patient stabilizes, after which oral treatment can begin. At discharge, the dose is lowered to 0,5 mg/kg once daily, with further gradual reductions as tolerated (see long-term treatment).

Other protocols, such as hydrocortisone constant rate infusion (CRI) (0,5 mg/kg/h) for adrenal crises, have been described in feline case reports. However, glucocorticoid choice and dosing largely depend on clinician preference, as evidence-based recommendations are lacking.[39]

Additional supportive treatment during hospitalization

In cats with gastrointestinal signs, gastro-protectants or antiemetics may improve recovery. Prophylactic antibiotics are not recommended and should be reserved for cases with a high risk of bacterial translocation and sepsis.

After initial stabilization, patients require regular reassessment. Fluid rates should be adjusted for dehydration and azotemia, and electrolytes monitored every 6–12 hours until balanced. Continuous electrocardiographic (ECG) monitoring is advised until arrhythmias resolve.

Table 5: Adjustment of DOCP dose according to the potassium concentrations on day 14 and 28 after DOCP injection.

Day 0	Day 14*/**	Day 28*/**	Recommended dose adaption	Special comment
Cats: DOCP 2,2 mg/kg SC	K normal	K normal	Inject DOCP at the same dose.	Recheck after 28 days, if this was after the first DOCP injection. Recheck after 3 months, if this was after the second DOCP injection.
	K normal	K low	No DOCP injection. Recheck electrolytes every 7 days until K is within the reference range. At that time inject DOCP, but reduce the dose by 10 % for every 7 days' delay. If potassium is within the reference range and sodium shows significant deviations, the dose adjustment on day 28 must also be based on the sodium concentration (e.g. if sodium is below the reference range, no decrease in the DOCP dose).	Recheck after 28 days.
	K normal	K high	Inject DOCP and increase dose by 10 %.	Recheck after 28 days.
	K low	K normal	Inject DOCP and decrease dose by 10 %. If potassium is within the reference range and sodium shows significant deviations, the dose adjustment on day 28 must also be based on the sodium concentration (e.g. if sodium is below the reference range, no decrease in the DOCP dose).	Recheck after 28 days.
	K low	K low	No DOCP injection. Recheck electrolytes every 7 days until K is normal. At that time inject DOCP but reduce the dose by 10 % for every 7 days' delay. If potassium is within the reference range and sodium shows significant deviations, the dose adjustment on day 28 must also be based on the sodium concentration (e.g. if sodium is below the reference range, no decrease in the DOCP dose).	Recheck after 28 days.
	K low	K high	Inject DOCP and increase dose by 10 %.	Recheck after 14 and 28 days.
	K high	K high	Inject DOCP and increase dose by (10-)20 %.	Recheck after 14 and 28 days.

*At each recheck, sodium and potassium should be measured. It is important always to measure the electrolytes in the same material (serum, heparin-plasma). Otherwise, the values cannot be compared.
**Sodium alone is less suitable for dose control than potassium. Ideally, sodium should be in the upper half of the reference range on day 14 and in the lower half of the reference range on day 28.

If adrenal crisis is suspected and emergency treatment is initiated promptly, cats can respond well to treatment. However, clinicians should be aware that in-hospital mortality at diagnosis has been reported to be higher in cats than in dogs (approx. 15 %).[39] This may reflect delayed diagnosis or greater difficulty in stabilizing feline patients. Compared to dogs, cats often require longer hospitalization. Reported median time to discharge in cats is approximately 4 days, with hospitalization durations of up to 10 days in moderately affected individuals and up to 13 days in more severely affected cases, whereas dogs are typically hospitalized for only 2–5 days.

Long-term treatment

Long-term treatment for cats with primary hypoadrenocorticism involves lifelong replacement of both mineralocorticoids and glucocorticoids.[44] Mineralocorticoid treatment should be started during hospitalization as soon as the cat is clinically stable and the danger of a too rapid increase in serum sodium has passed. Two formulations of mineralocorticoids are available, one long-acting (desoxycorticosterone pivalate), and a short-acting (fludrocortisone). The most commonly used medication for substituting glucocorticoids is prednisolone.

It is important to emphasize that, initially, frequent follow-up evaluations are required to determine the optimal mineralocorticoid and glucocorticoid dosages for each individual cat. Thereafter, regular lifelong monitoring remains essential, as dosage adjustments may be necessary over time.

Mineralocorticoid treatment

Desoxycorticosterone pivalate (DOCP)

DOCP is a long-acting synthetic mineralocorticoid. Due to the absence of both 11β- and 17α-hydroxylation, it is expected to possess minimal or no glucocorticoid activity. It is supplied as an extended-release suspension in two products registered only for dogs – Percorten-V® (Elanco US Inc., Greenfield, Indiana, USA) and Zycortal® (Dechra Veterinary Products GmbH, Aulendorf, Germany) – with only Zycortal® authorized within the European Union and Switzerland. Although neither formulation is licensed for cats, both are routinely employed in feline practice.

The authors' preferred DOCP starting dose in cats is 2,2 mg/kg subcutaneously, every 28 days. This is higher than the typical starting dose in dogs, as cats appear to have a greater mineralocorticoid requirement.[44] Approximately half of feline patients will require a dose increase within the first few months, and some may also need a shortened injection interval of 21–25 days.[44] Conversely, other cats may eventually tolerate gradual dose reductions. Long-term maintenance doses typically range from 1,3 to 3,0 mg/kg, with many cats stabilizing around 2,0–2,3 mg/kg every 28 days.[39,44]

After the first injection, serum electrolytes should be re-evaluated at 14 and 28 days, with dose modification guided by absolute sodium and potassium concentrations (the Na^+/K^+ ratio is not recommended by the authors). Adjustments of the DOCP dose can be made according to Table 5. Subsequent rechecks are typically done after 28 days, unless the DOCP dose requires adjustment, in which case Table 5 should be consulted again. The 14-day check confirms initial response and informs the next dose, while the 28-day visit permits fine-tuning of both dose and interval.

Once the appropriate DOCP dose has been determined, regular rechecks every 4–6 months, aligned with the scheduled DOCP injections, are recommended. A well-controlled cat should exhibit an active and happy demeanor, have a healthy appetite, and show no signs of overtreatment, such as excessive weight gain, lethargy, muscle wasting, dull hair coat, hair loss, polyuria and polydipsia. Serum potassium and sodium concentrations should be within the reference interval. Although cat owners can be instructed on how to perform the injection at home, it is usually administered by the veterinarian, as, in contrast to dogs, most owners prefer not to give it themselves.

Fludrocortisone acetate

Fludrocortisone acetate, a short-acting mineralocorticoid, not only demonstrates significant mineralocorticoid activity but also possesses notable glucocorticoid activity, estimated to be approximately 10 times that of cortisol. It is available in two oral formulations (Florinef®, Viatris Pharma GmbH, Steinhausen, Switzerland and Astonin®H, Merck Healthcare GmbH, Weiterstadt, Germany; both human products and not registered for cats) and can serve as an alternative to DOCP for supplementing mineralocorticoids.

While treatment with short-acting formulations may be more cost-effective in some countries, the majority of reported cases now use DOCP since its registration in the EU and other countries worldwide.

The recommended starting dose of fludrocortisone is 0,01–0,02 mg/kg/day administered orally, divided into two daily doses. The effectiveness of fludrocortisone acetate treatment is regularly evaluated by monitoring clinical manifestations, renal function (blood urea nitrogen and serum creatinine concentration), and serum electrolyte concentrations (sodium and potassium). Adjustment of the fludrocortisone acetate dosage is primarily guided by serum potassium concentrations. In a larger case series, the median final dosage at the end of the follow-up period was 0,03 mg/kg/day, with a reported range of 0,01–0,05 mg/kg/day.[39] The main limitation of fludrocortisone acetate is its significant, yet unpredictable, glucocorticoid activity. However, unlike in dogs, this effect appears to be less pronounced in cats. As in dogs, it may become necessary to switch from fludrocortisone to DOCP if serum electrolyte concentrations cannot be adequately controlled.

Glucocorticoid treatment

Daily glucocorticoid supplementation is essential for cats receiving DOCP. At discharge, prednisolone is typically prescribed at 0,5–1,0 mg/kg/day (median ≈ 0,7 mg/kg/day). After the first week – or by the 14-day recheck – the dose can often be reduced to 0,25–0,5 mg/kg/day, depending on the cat's clinical condition.

Long-term maintenance doses should be individualized based on clinical response, with most cats stabilizing between 0,08 and 0,5 mg/kg/day (median ≈ 0,3 mg/kg/day).[44] Reported extremes range from 0,02 to 2 mg/kg/day.[39] The goal is to maintain stability without signs of glucocorticoid excess. No laboratory marker, including endogenous ACTH, has yet been validated for routine adjustment of glucocorticoid dosing in feline replacement therapy.

In cats that are difficult to medicate orally, a liquid prednisolone preparation may be a suitable alternative. If oral administration is not feasible, monthly methylprednisolone injections at a dose of 10–20mg/cat/month may be used. However, short-acting glucocorticoids are preferred, as long-acting forms increase diabetes risk and should be reserved as a last resort when oral treatment is impossible, and euthanasia is being considered.

As data on whether fludrocortisone acetate alone provides sufficient glucocorticoid activity are currently lacking, the authors use the same glucocorticoid preparations and doses in cats receiving fludrocortisone as in those treated with DOCP. Given that cats generally require higher glucocorticoid doses than dogs, it is assumed that discontinuation of additional glucocorticoid supplementation is less often feasible in cats compared to dogs.

Glucocorticoid requirements during stressful situations:

The need for glucocorticoids rises during stress and may require temporary dose adjustments based on the maintenance dose, stress perception, and living conditions. Owners should receive initial training and must seek veterinary advice in acute situations. Generally, the daily dose can be doubled or tripled for mild stress (e.g., travel, visitors), and transiently increased to 0,5–2 mg/kg/day during illness, trauma, or before surgery. Care must be taken to avoid frequent or excessive increases, as prolonged overdosing can cause iatrogenic hyperadrenocorticism, which may manifest with clinical signs such as excessive weight gain, polyphagia, lethargy, polyuria and polydipsia. Moreover, due to glucocorticoid-induced insulin-resistance, cats have an increased risk of developing diabetes mellitus.

Prognosis

Cats with hypoadrenocorticism that survive initial hospitalization have a favorable long-term prognosis.[39,44] In the largest cohort reported to date, the median all-cause survival time was about 5,5 years, and a specific median for adrenal-related deaths could not be reached because most cats either remained alive or died of unrelated disease.[39] One-year survival attributable to hypoadrenocorticism approached 75 %, and survival did not differ significantly among cats treated with DOCP, fludrocortisone, or prednisolone alone in cats with eunatremic and eukalemic hypoadrenocorticism.[39] Among 39 documented cases, only seven cats were euthanized, most within the first week of hospitalization owing to a poor initial response or to serious comorbidities such as lymphoma, lymphocytic panhypophysitis, or newly developed diabetes mellitus.[35] Cats without major concurrent disease and those presenting with a more chronic course tended to do well, and the longest recorded survival exceeded six years. Owner compliance also influences outcome. In a group of eight cats followed for up to 70 months on DOCP, six remained stable long-term; the two early losses were linked to financial constraints or difficulty administering prednisolone rather than a treatment failure itself.[44] Therefore, as most cats are even more difficult to medicate on a daily basis for the rest of their life than dogs, it is crucial to emphasize to owners the importance of lifelong, consistent medication administration and regular veterinary monitoring. Deviation from the prescribed DOCP medication schedule, including skipping doses or extending the treatment interval without veterinary authorization, can significantly elevate the risk of a crisis. Consequently, a high level of commitment is required of the owner. Overall, when regular monitoring and medication can be maintained, most cats enjoy a good quality of life and survive for several years after diagnosis.

Hypoadrénocorticisme félin: nouvelles perspectives sur les signes cliniques, le diagnostic et le traitement

L'hypoadrénocorticisme chez le chat est un trouble endocrinien rare qu'il convient d'envisager chez les patients présentant des signes cliniques vagues, intermittents ou fluctuants. Des signes gastro-intestinaux chroniques, un retard de croissance ou des épisodes de choc hypovolémique doivent éveiller la suspicion clinique. Les chats British Shorthair pourraient être surreprésentés parmi les chats atteints et les cliniciens doivent être particulièrement vigilants lorsque cette race présente des signes compatibles.

Le diagnostic définitif nécessite un test de stimulation à l'ACTH, qui doit être réalisé avant le début de tout traitement aux glucocorticoïdes afin d'éviter toute interférence diagnostique. Chez les chats cliniquement stables, la mesure de la cortisolémie de base peut servir d'outil de dépistage initial. Cependant, comme aucun seuil spécifique aux félins n'a été validé, la valeur seuil couramment utilisée de 2 µg/dL (55 nmol/L) pour exclure l'hypoadrénocorticisme doit être interprétée avec prudence, car elle peut être moins fiable que chez les chiens.

La prise en charge à long terme implique un traitement substitutif à vie par glucocorticoïdes, généralement sous forme de prednisolone par voie orale. Les chats nécessitent généralement des doses d'entretien plus élevées que les chiens pour maintenir une stabilité clinique. La déficience en minéralocorticoïdes est traitée par l'administration sous-cutanée d'une préparation de désoxycorticostérone à libération prolongée (Zycortal®) administrée mensuellement à une dose initiale de 2,2 mg/kg – nettement supérieure aux doses canines. À titre d'alternative, l'acétate de fludrocortisone (Florinef® ou Astonin®H) peut être administré par voie orale à raison de 0,01 à 0,02 mg/kg/jour dans certains cas. Une surveillance régulière des taux de sodium et de potassium est essentielle pour ajuster les doses de minéralocorticoïdes et optimiser le traitement.

Les chats atteints étant incapables de mettre en place une réponse adéquate au stress, une supplémentation supplémentaire en glucocorticoïdes («boost glucocorticoïde») est nécessaire lors de situations stressantes ou en cas de maladie concomitante. Les propriétaires doivent être informés des signes de crise surrénale, qui peuvent être plus subtils et difficiles à reconnaître chez les chats que chez les chiens. L'accent doit être mis sur l'administration régulière des médicaments et un suivi vétérinaire de routine afin d'assurer le contrôle à long terme de la maladie et la qualité de vie.

Mots clés: glande surrénale, stimulation à l'ACTH, cortisol, glucocorticoïde, hyperkaliémie, minéralocorticoïde

Ipoadrenocorticismo felino: nuove prospettive su caratteristiche cliniche, diagnosi e trattamento

L'ipoadrenocorticismo è un'endocrinopatia rara nei gatti, che dovrebbe essere presa in considerazione in presenza di sintomi clinici aspecifici, intermittenti o ricorrenti. Nei casi di disturbi gastrointestinali cronici, mancato aumento di peso o episodi ricorrenti di shock ipovolemico, l'ipoadrenocorticismo dovrebbe essere incluso tra le diagnosi differenziali. I gatti di razza British Shorthair potrebbero essere sovrarappresentati, pertanto in questa razza è necessaria particolare attenzione in presenza di sintomi compatibili.

La diagnosi definitiva richiede un test di stimolazione con ACTH, che deve essere eseguito prima dell'inizio della terapia con glucocorticoidi per evitare interferenze diagnostiche. Nei gatti clinicamente stabili, la misurazione del cortisolo basale può essere utilizzata come test di screening iniziale. Tuttavia, poiché non sono ancora stati validati valori soglia specifici per il gatto, il valore comunemente utilizzato per escludere l'ipoadrenocorticismo di 2 µg/dL (55 nmol/L) deve essere interpretato con cautela, in quanto potrebbe essere meno affidabile rispetto al cane.

La terapia a lungo termine prevede una sostituzione glucocorticoidea per tutta la vita, generalmente con prednisolone somministrato per via orale. Rispetto ai cani, i gatti richiedono solitamente dosi di mantenimento più elevate per raggiungere una stabilità clinica. Il deficit di mineralocorticoidi viene trattato con un'iniezione sottocutanea mensile del preparato depot desossicorticosterone (Zycortal®) a una dose iniziale di 2,2 mg/kg; tale dose è significativamente più alta rispetto a quella raccomandata per il cane. In alternativa, si può utilizzare la somministrazione orale quotidiana di fludrocortisone acetato (Florinef® o Astonin® H) alla dose di 0,01–0,02 mg/kg/die. Il monitoraggio regolare dei livelli di sodio e potassio è essenziale per adattare individualmente la dose di mineralocorticoidi e ottimizzare la terapia.

Poiché i gatti affetti non sono in grado di sviluppare una risposta adeguata allo stress, durante situazioni stressanti o malattie concomitanti è necessaria una somministrazione aggiuntiva di glucocorticoidi ("boost" glucocorticoideo). I proprietari devono essere adeguatamente informati sui segni di una crisi surrenalica, che nei gatti può essere più subdola e difficile da riconoscere rispetto ai cani. Una somministrazione costante dei farmaci e controlli veterinari regolari sono fondamentali per garantire un buon controllo della malattia e una qualità di vita soddisfacente.

Parole chiave: Surreni, stimolazione ACTH, cortisolo, glucocorticoidi, iperkaliemia, mineralocorticoidi

Feline hypoadrenocorticism: Emerging Insights into Clinical Features, Diagnosis, and Treatment

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