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Hypoadrenocorticism in a Kitten

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Abstract

This case describes how a 7-month-old, female, intact kitten was diagnosed with hypoadrenocorticism and fully recovered after treatment with fludrocortisone acetate. The cat showed signs of weight loss, severe weakness, and anorexia. Clinical findings included severe dehydration, lethargy, and moderate hypothermia. Blood examinations showed severe azotemia, hypernatremia, hypochloremia and hyperkalemia. Hypoadrenocorticism was diagnosed on the basis of low cortisol concentration during hospitalization. The cat had a full recovery after being treated with on daily dosage of fludrocortisone acetate and prednisolone; and is still well after one year. We believe this is the first case describing hypoadrenocorticism in a kitten younger than 12 months. This case demonstrates the success of fludrocortisone acetate as the treatment, using the level of cortisol concentration as an index; and that evaluating the cortisol concentration is a good method to monitor the change of hypoadrenocortism; and that hypoadrenocorticism could be reversed with a good treatment.

Keywords: *Cortisol, Feline, Fludrocortisone acetate, Hypoadrenocorticism, Kitten*

Introduction

Hypoadrenocorticism, also known as Addison's disease (AD), is a severe or total deficiency of cortisone. AD is well-described in dogs. Its estimated prevalence is 0.3% to 1.1% [1] and is generally diagnosed at the age between 3 months and 14 years [2,3]. Confirmation of the AD diagnosis is often by ACTH stimulating test and its reading of the cortisol levels in blood [2,4-6]. However, testing the basal plasma cortisol level could be an easier, more reliable and less costly method than ACTH stimulating test [7]. AD is rarely reported in cats. Up to date, approximate 40 cases have been reported [8-18]. In cats, primary AD is less likely to be found, even rarer in cats younger than 12 months. The majority of patients are shorthair domestic cats [8,11,13]; and the onset age is between 1.5 to 14 years old (median 5 years old) [13]. There is no clear evidence showed the morbidity with sex, age and breeding in cats [5]. Some case reports showed that a few factors, such as corticosteroid or/and megestrol acetate withdrawal, neoplastic infiltration, immune-mediated problem, and trauma could contribute to primary or secondary AD [8,14,15,19].`

Basically, an excellent outcome of AD can be achieved by using medicines, with a post-diagnosis life expectancy of up to 70 months [20]. There are two protocols to treat AD, using either the combination of methyprednisolone and DOCP or the combination of fludrocortisone acetate and prednisone/prednisolone [2,5,13]. Reports stated a consecutive treatment by using the second protocol can keep the cats alive over than one year without clinical signs [12,13,16,18]. The case aims to describe an AD occurred in a very young kitten and the good outcome after diagnosis and treatment as, based on the authors' knowledge, no report has described AD in a kitten age under 1 year old.`

Case Description

A 7 months old, female intact, and mixed breed shorthair cat was referred to a small animal clinic with a body weight of 1.6 kg and a body condition score of 3/9. The owner said the kitten showed progressive lethargy, unwilling to move, intermittent vomiting and diarrhea 3 weeks ago. The cat had no record of using exogenous steroid or mestrol acetate. After performing the physical examination (Day 0), the patient also showed clinical signs of progressive weight loss, hypothermia (36.5°C), anorexia, 10%-12% dehydration, and mucous membranes were pale and dry. Thoracic auscultation, abdomen palpation and neurological examination, blood pressure, heart rate, and respiratory rate were within normal range. The FIV/FeLV/FCoV (Speed Trio, Virbac) and the feline distemper kit (FPV Ag Test Kit, Bionote Anigen) displayed negative results.`

Serum biochemistry revealed electrolyte abnormalities, severe azotaemia, and dehydration (Tables 1 and 2). Urinalysis showed isosthenuria (specific gravity 1.011), and mild proteinuria (1+). Abdominal radiography and ultrasonography returned no specific finding. Based on above findings, taking account of especially the values of electrolytes, this case could be either acute kidney injury (AKI) or AD.`

On Day 0, the initial treatments included intravenous (IV) fluid therapy. The cat received 100 mL of 0.9% normal saline subcutaneously and 0.9% normal saline was administered at 120 mL/kg/day to correct the dehydration. Two mL of 10% calcium-gluconate IV and 1 IU of regular insulin SC were administered as a cardioprotective agents because of the severe hyperkalemia. To avoid hypoglycemia, 1 mL of 10% glucose was injected by IV slowly. 200 mg lanthanum carbonate was given twice daily PO for hyperphosphatemia. Eight hours later, the

Table 1: Serial monitoring of hematological parameters in the kitten.

Parameter	unit	Day 0	Day 2	Day 8	Day15	Day21	Day 213	RI
RBC	M/μL	9.98	7.26	7.82	5.58	5.68		6.54-12.20
HCT	%	36.1	27.0	29.2	21.0	23.5	25.6	30.3-52.3
HGB	g/dL	13.3	11.4	10.3	7.6	7.7	8.8	9.8-16.2
MCV	fL	36.2	37.2	37.3	37.6	41.4	41.0	35.9-53.1
MCH	pg	13.3	15.7	13.2	13.6	13.6	14.1	11.8-17.3
MCHC	g/dL	36.8	42.2	35.3	36.2	32.8	34.4	28.1-35.8
RDW	%	29.0	24.9	29.9	25.5	33.8	22.4	15.0-27.0
RETIC	K/μL	3.0	2.9	25.8	4.5	67.6	5.6	
RETIC-HGB	pg			15.7	14.8			3.0-50.0
WBC	K/μL	12.02	17.01	11.70	11.20	8.46	10.48	2.87-17.02
NEU	K/μL	9.65	12.49	10.16	7.38	4.92	5.34	1.48-10.29
LYM	K/μL	1.98	3.38	0.92	3.03	2.26	3.14	0.92-6.88
MONO	K/μL	0.31	0.29	0.54	0.33	0.70	0.25	0.05-0.57
EOS	K/μL	0.01	0.27	0.00	0.35	0.44	1.67	0.17-1.57
BASO	K/μL	0.07	0.58	0.08	0.11	0.14	0.08	0.01-0.26
PLT	K/μL	574	272	813	285	994	275	151-600
MPV	fL	18.2	17.8	16.7	16.9	16.2	17.6	11.4-21.6

RBC: Red Blood Cell; HCT: Haematocrit; HGB: Hemoglobin; MCV: Mean Cell Volume; MCH: Mean Cell Hemoglobin; MCHC: Mean Corpuscular Hemoglobin Concentration; RDW: Red Cell Distribution Width; RETIC: Reticulocyte; RETIC-HGB: Reticulocyte Hemoglobin; WBC: White Blood Cell; NEU: Neutrophil; LYM: Lymphocyte; MONO: Monocyte; EOS: Eosinophil; BASO: Basophil; PLT: Platelet; MPV: Mean Platelet Volume.

Table 2: Serial monitoring of the serum biochemistry in the kitten.

parameter	**Unit**	**initial**	**8 h**	**Day 1**	**Day 2**	**Day 3**	**Day 10**	**Day 13**	**Day 17**	**Day 21**	**Day 22**	**Day 23**	**Day 24**	**Day 25**	**Day 26**	**Day 213**	**RI**
Glucose	mg/dL	227	44	141	156		161		148							116	74-159
Total protein	g/dL	10.3			8.1		10.2		7.2	7.9	7.2	7.5	6.7	7.5		7.2	5.7-8.9
Albumin	g/dL	4.1			3.8		4.5		3.4							3.2	2.2-4.0
BUN	mg/dL	>130		62	40		110	42	37	42	30	28	26	31		41	16-36
Creatinine	mg/dL	Over		1.9	1.7		Over	1.5	1.5	2.5	1.9	1.9	2.2	1.9		3.3	0.8-2.4
ALT	U/L	13			80		22		25								12-130
ALP	U/L				17		25		18								14-111
Inorganic phosphorus	mg/dL	14.0		6.3	5.4		>16.1	6.0	5.4					6.8			3.1-7.5
Calcium	mg/dL	10.9							10.0								7.8-11.3
Potassium	mmol/L	8.1	4.7	7.1	4.4	4.3	7.5	4.3	3.5	4.4	4.1	4.4	4.1	4.0	5.3	4.7	3.5-5.8
Sodium	mmol/L	133	140	143	148	146	132	160	160	153	154	156	154	155	162	158	150-165
Sodium/potassium		16	30	20	34	34	18	37	45	35	38	35	37	39	31	34	
Chlorine	mmol/L	98	106	108	109	111	100	123	120	117	117	119	119	116	118	127	112-129

BUN: Blood Urea Nitrogen; ALT: Alanine Aminotransferase; ALKP: Alkaline Phosphatase.

potassium concentration had become normal. After another injection of 1 mL of 10% glucose, the patient's appetite, spirit started to improve at midnight, and its body temperature gradually returned to 38.4°C. On Day 1, the potassium concentration increased again. The order was the same as Day 0. On Day 2, the potassium and Inorganic phosphate returned to the normal range so cardioprotective agents and lanthanum carbonate were stopped. The kitten presented polyuria/polydipsia. The haematological results showed mild anemia. The BUN was slightly higher and the electrolytes (sodium and chloride) were slightly lower. The other biochemical values were normal. At this point, the kitten had been rescued from emergency situation.`

Consequently, IV fluid was changed to 60 mL of 0.9% saline subcutaneous once daily, On Day 3, the owner asked the kitten to be discharged. We prescribed the same treatment as Day 2. One week later, on Day 10, the patient's condition worsened again and was re-hospitalized. It showed anorexia, lethargy, and severe dehydration. Based on the previous data, we considered the possibility of a rare disease, Addisonian crisis. Haematological and biochemical findings showed mild non-regenerative anemia, severe azotemia, and electrolytes abnormal (Tables 1 and 2). The cat was administered 0.9% normal saline at 120 mL/kg/day and dosed 0.02 mg/kg fludrocortisone acetate and 0.5 mg/kg prednisolone oral once daily as mineralocorticoid and glucocorticoid supplements. On Day 15, the value of cortisol concentration (1.1 ug/dL) became lower than the reference value. On Day 26, the value of cortisol concentration came up to the normal range (2.7 ug/dL) so we decided to tapered and then stopped the oral drugs. On Day 332, the patient was spayed and its cortisol concentration was normal (2.7 ug/dL) (Table 3). The cat has been very well since its discharged a year ago.`

Table 3: Serial monitoring of the serum cortisol concentration.

parameter	unit	Day 15	Day 26	Day 332	RI
cortisol	μg/dL	1.1	2.7	2.7	1.7-4.2

Discussion

We believe this is the first report case of AD in an intact kitten younger than 1 years old. A report stated that the disease all occurred in neutered adults cats over than 1 years old and the mean age was 5.8±3.7 and the range was between 1.5 and 14 [13]. Some researchers have stated that this disease can occur in 1 years old, neutered cats [10], in 3-6 years old cats [12,16,18] and in cats older than 8 years old [14,15,21,22]. Our study advances the knowledge that AD can occur in an intact kitten younger than 1 year old.`

Whether to stop fludrocortisone acetate after the clinical signs have disappeared has been a controversial issue. Some researchers believe treatment of AD is lifelong because it cannot be reversed [2,12,16,18,23]. A report mentioned that a cat suffered from AD again after the treatment was changed from twice daily to once daily [12]. In fact, the decision of prescribing a consecutive treatment is based on how good the clinical signs and electrolyte are [2,18,23]. One case pointed out the cortisol concentration became to normal 40 days after the AD treatment by using prednisolone was stopped [21]. We believe to use fludrocortisone acetate could achieve the same result as the last case based on the change of the basal cortisol concentration in our case during the treatment. After Day 26, the cortisol concentration and other biochemical values have been normal. Treatment on the kitten stopped after Day 26. On Day 332, the cortisol concentration was still normal, indicating that the kitten had recovered from Addisonian crisis. The mechanism behind the kitten's recovery from AD is unknown.`

In conclusion, this is the first case which describes AD in a kitten younger than 12 months old. Secondly, the case shows that evaluating the values of cortisol concentration is a good method to monitor the change of AD. Thirdly, fludrocortisone acetate is also a good method to treat AD. And finally, AD could be reversed after a good treatment.`

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